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Health Care Cost Containment Measures and Mortality in Hennepin County's Medicaid Elderly and All Elderly

GREGORY L. LINDBERG, MA, NICOLE LURIE, MD, MSPH, SUSAN BANNICK-MOHRLAND, MD,
ROBERT E. SHERMAN, PHD, AND PAUL A. FARSETH, BA

Abstract: In Minnesota, several health care cost containment measures occurred about the time Medicare's Prospective Payment System (PPS) was implemented. These included a moratorium on additional nursing home beds, preadmission screening of nursing home applicants, and rapid growth in HMO (health maintenance organization) enrollment by Medicare recipients. Hospital days per elderly Medicaid recipient decreased by 38 percent for those in nursing homes and by 35 percent for those not in nursing homes from 1982 to 1984. By 1986, hospital days per recipient had decreased 53 and 55 percent, respectively, from the 1982 level.

Age-adjusted mortality rates for elderly Medicaid nursing home residents for the period 1977 through 1986 showed an increasing trend after 1982. Estimated age-adjusted mortality rates for the entire County population, which had decreased steadily from 1970 to 1982, rose significantly above the projected rate in 1984, 1985, 1986, and 1987.

We conclude that, coincident with the institution of the PPS and other health care cost containment measures, use of hospital care has fallen for all elderly Medicaid recipients, age-adjusted mortality rates among those in nursing homes have increased, and the mortality rate trend for the total Hennepin County elderly population has stopped declining. (*Am J Public Health* 1989; 79:1481–1485.)

Introduction

Medicare's transition to a system of "fixed price" hospital reimbursement based on diagnosis-related groups, the prospective payment system (PPS) has triggered concerns about the consequences, particularly the chance that the poor and the chronically ill may be at increased risk of underservice.[1] At some point there is trade-off between cost and quality of care. Two manifestations of this trade-off might be an increase in age-adjusted mortality rates for Medicare patients and signs of premature hospital discharge to nursing homes.

In a cross-sectional area study of Medicare spending levels, Hadley, found that greater Medicare spending per beneficiary is associated with significantly lower mortality rates.[2] He also reported that national mortality rates for those age 65 and older have either changed little or actually increased since 1982, after declining throughout the 1970s. In Wisconsin, Sager, *et al*, reported a shift in the site of death from the hospital to the nursing home and an increase in the age-specific death rate among elderly nursing home residents from 1982 to 1985.[3] Comparing 12-month periods immediately before and after the institution of the PPS, they found a 72 percent increase in the rate of hospitalization and a 26 percent decline in hospital length of stay among elderly Medicaid nursing home patients. In a later study, Sager, *et al*, found that shifts in the location of death from hospitals to nursing homes were most pronounced in states with large declines in hospital length of stay and high population enrollments in health maintenance organizations (HMOs).[4] In a survey of nursing homes in Portland, Oregon, Lyles[5] found a 26 percent increase in the age-adjusted mortality rate among nursing home residents from 1983 to 1984. Lewis, *et al*,[6] reported that Medicare patients discharged for the first time to skilled nursing facilities in 1984 were more likely to be bed-confined, comatose, or have indwelling catheters than those discharged in 1980; however, these trends began before the institution of the PPS.

Meyer-Oakes, *et al*,[7] found a significant reduction in length of hospital stay but no change in hospital mortality post-PPS. However, they did not examine the possibility of increased post-discharge mortality. Shortell and Hughes, using 1983–84 data from 981 hospitals in 45 states found that the ratio of actual to predicted death rates for Medicare recipients with one of 16 selected diagnoses was 6 to 10 percent higher in those states with the most stringent rate reviews and 5 to 6 percent higher in those with the strictest procedures for reviewing applications for certificate of need.[8]

Fitzgerald, *et al*,[9] reported an increase in short- and long-term admissions to nursing homes after PPS for hip fracture patients in Indianapolis. Linn, *et al*,[10] have reported an increase in complication rates in patients undergoing head and neck cancer surgery after implementation of PPS.

The PPS was implemented in Hennepin County, Minnesota (the greater Minneapolis area) over a period of several months beginning in October 1983. Roughly coinciding with the implementation was the imposition of a statewide moratorium on new nursing home beds. State and county efforts to divert nursing home applicants through preadmission screening and alternative care grants were phased in beginning in 1982 and were fully implemented by 1986. The number of nursing home beds in Hennepin County has remained relatively stable over the past 10 years at around 12,000 and there has been a major increase in recent years in the provision of home health care services. For example, expenditures in Hennepin County under the state and county funded Alternative Care Grant Program increased from $200,000 in 1983 to $5 million in 1988. This represents only a small portion of the total public and private expenditures for these services. In addition, since the end of 1984, Medicare enrollees have been shifting from fee-for-service to prepaid, capitated care. As of December 31, 1986, 47 percent of all Twin Cities Metropolitan Area residents age 65 or older were enrolled in HMOs (Minnesota Department of Health, personal communication).

The present study describes changes in hospital use and

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age-adjusted mortality rates that have occurred in Hennepin County following the implementation of these policy initiatives. Examined are: hospital utilization by elderly (65 and older) Medicaid recipients; the age-adjusted mortality rate for the elderly Medicaid caseload; and the age-adjusted mortality rate trend for the total elderly population in Hennepin County. The Medicaid caseload was selected for analysis because it represents a vulnerable population that is of special concern to state and local government and because health care utilization data were readily available through the Minnesota Department of Human Services. Members of this caseload differ from the general elderly population in that they are more likely to be from low income groups, may be sicker, and are more likely to have used extensive medical care or nursing home services.

Methods

Hospital Utilization Data

Data on hospital utilization by institutionalized elderly Hennepin County Medicaid recipients were obtained from the Minnesota Medicaid Information System. The Minnesota Department of Human Services provided research files containing annual client eligibility summaries and hospital payment records for the years 1980 through 1986. These included all Medicare claims in which Medicaid was a financial participant. The years, 1980 through 1982, represent a period unaffected by the PPS or local cost containment policies.

Recipients were categorized as institutionalized or noninstitutionalized based on their living arrangement codes. The living arrangement code indicated the recipient's status at the last eligibility review or living arrangement change before the end of each year. Those in nursing homes or state hospitals were considered institutionalized while others were considered noninstitutionalized. During the years examined, the number of elderly institutionalized Medicaid recipients ranged from 6,800 to 7,348, and the number of elderly noninstitutionalized recipients ranged from 3,630 to 3,907. Hospital utilization and mortality rates were calculated for the full population of elderly recipients each year. There have been no major changes in Medicaid eligibility criteria or procedures from year to year that would have significantly altered the character of the caseload.

An additional analysis examined changes in hospital utilization during the last six months of life among elderly Medicaid recipients who died. Recipients selected for this analysis were those whose cases closed because of death during the last half of the year, and who had at least six continuous months of eligibility preceding their death.

The tabled hospital utilization data are presented with estimated 95 percent confidence intervals. The percentage differences in utilization between years are also presented in the text with 95 percent confidence intervals.

Mortality Data

Mortality among the Medicaid caseload was determined from annual Medicaid client eligibility files based on case closings in which death was coded as the reason for closing. To identify mortality trends over the longer term, these files were examined for 1977, the earliest year available, through 1986. Age-specific mortality rates for institutionalized and noninstitutionalized elderly Medicaid recipients were computed by five-year age intervals for each year. Age-adjusted mortality rates were then computed for these groups using, as a standard population, the mean of the age distributions for the first and last years of the time series (1977 and 1986).[11]

Age-adjusted mortality rates for the entire elderly population of Hennepin County (both Medicaid and non-Medicaid) for the period 1970–87 were derived using the death certificate data maintained by the Minnesota Department of Health and age-specific population projections developed for Hennepin County.[12] Death certificate data in Hennepin County are considered accurate and complete, and the population projections for the elderly population utilized a cohort-component procedure which is thought to result in estimation accurate to one percent or so over the study time frame. (Population estimates for the 1970–80 period were developed through linear interpolation of census data.)

Linear regression on logarithmicly transformed data was used to estimate the age-adjusted mortality rate trend during the period preceding the implementation of cost containment measures (i.e., prior to 1983). Subsequent age-adjusted mortality observations were examined to see if they fell within the 95 percent confidence interval (for observations) of the pre-cost containment trend.

Results

Changes in Hospital Utilization

There was a marked reduction in hospital days per Medicaid recipient beginning in 1983. This is due not only to a substantial reduction in average hospital length of stay, but also a large decrease in hospitalization rates (Table 1). From 1982 to 1984 (the years before and after implementation of the PPS) hospital days per Medicaid recipient per year fell 38 percent (95% CI = 32.5, 43.2) for institutionalized and 35 percent (95% CI = 26.8, 43.5) for noninstitutionalized recipients.

Hospitalization rates continued to decrease from 1984 to 1986 for both institutionalized and noninstitutionalized recipients. Length of stay decreased further as well, but at a lesser rate. The total reductions in hospital days per person for the four-year period from 1982 to 1986 were 53 percent (95% CI = 48.9, 57.6) and 55 percent (95% CI = 48.2, 62.6) for institutionalized and non-institutionalized recipients, respectively.

We also examined the change in hospital use among those recipients who died. We hypothesized that those who died would, because of the severity of their illness, experience a smaller proportionate reduction in hospital care than the general caseload. The results shown in Table 2 indicate that, from 1982 to 1984, decedents experienced percentage reductions in hospitalization that were similar to those for the general elderly Medicaid population. Total average hospital days during the last six months of life decreased by 41 percent (95% CI = 28.4, 54.6) for institutionalized decedents and by 39 percent (95% CI = 16.3, 61.7) for noninstitutionalized decedents. From 1984 to 1986, there was no significant change in this measure.

Changes in Mortality Rates

Figure 1 presents the age-adjusted mortality rate for institutionalized elderly Medicaid recipients in Hennepin County for 1977–86. The mortality rate shows an increasing trend, although the number of pre-1983 observations is small and large fluctuations that occurred in 1978 and 1979 appear out of character with other year-to-year changes. An examination of the age-adjusted mortality rates for the remainder of the state's institutionalized Medicaid caseload showed that identical fluctuations occurred in all areas of the state during

TABLE 1—Hospital Utilization among Medicaid Recipients Age 65 and Older

	Year						
	1980	1981	1982	1983	1984	1985	1986
Institutionalized							
Number of Recipients	6800	7070	6996	7122	7296	7348	7239
Admissions per year per 1000 recipients	361 (343.2, 378.8)	379 (397.2, 360.8)	387 (369, 405)	356 (338.6, 373.4)	322 (304.9, 339.1)	297 (281.1, 312.9)	258 (244.5, 271.5)
Mean LOS per admission	12.2 (11.75, 12.65)	11.5 (11.09, 11.91)	11.3 (10.87, 11.73)	9.7 (9.33, 10.07)	8.4 (8.09, 8.71)	8.3 (7.93, 8.67)	7.9 (7.57, 8.23)
Hospital days per recipient per year	4.4 (4.13, 4.67)	4.4 (4.14, 4.66)	4.4 (4.14, 4.66)	3.5 (3.72, 3.28)	2.7 (2.52, 2.88)	2.5 (2.32, 2.68)	2.0 (1.84, 2.16)
Non-institutionalized							
Number of Recipients	3817	3907	3632	3630	3776	3846	3630
Admissions per year per 1000 patients	496 (467, 525)	475 (446.8, 503.2)	538 (506.8, 569.2)	468 (438.8, 497.2)	415 (388.5, 441.5)	340 (316.5, 363.5)	293 (271, 315)
Mean length of stay per admission	11.2 (10.6, 11.8)	10.5 (9.9, 11.1)	10.1 (9.4, 10.8)	8.8 (8.3, 9.3)	8.5 (7.8, 9.2)	7.8 (7.4, 8.2)	8.3 (7.3, 9.3)
Hospital days per Recipient per year	5.6 (2.07, 2.73)	5.0 (4.59, 5.41)	5.4 (4.97, 5.83)	4.1 (3.75, 4.45)	3.5 (3.15, 3.85)	2.6 (2.34, 2.86)	2.4 (2.07, 2.73)

95% Confidence intervals appear in parentheses.

TABLE 2—Hospitalization Utilization among Medicaid Decedents Age 65 and Older during the Last Six Months of Life

	Year						
	1980	1981	1982	1983	1984	1985	1986
Institutionalized							
Number of Decedents	474	512	550	546	615	569	596
Approximate number of Admissions per 6 months/1000 decedents*	572 (560, 635)	607 (545, 669)	573 (514, 632)	476 (422, 530)	452 (402, 502)	415 (365, 465)	443 (393, 493)
Approximate mean length of stay	13.1 (11.5, 14.7)	12.1 (10.8, 13.4)	11.3 (N/A)**	9.9 (N/A)**	8.4 (7.5, 9.3)	9.0 (7.7, 10.3)	8.7 (7.7, 9.7)
Hospital Days per Recipient in final 6 months	7.5 (6.3, 8.7)	7.3 (6.2, 8.4)	6.5 (5.5, 7.5)	4.7 (3.8, 5.6)	3.8 (3.2, 4.4)	3.7 (2.9, 4.5)	3.8 (3.1, 4.5)
Non-institutionalized							
Number of Decedents	48	65	75	58	78	49	51
Approximate number of Admissions per 6 Months/1000 decedents*	1354 (1079, 1629)	1015 (805, 1225)	1240 (1028, 1452)	963 (760, 1206)	1115 (922, 1308)	878 (639, 1117)	1196 (948, 1444)
Approximate mean length of stay	14.8 (10.6, 19)	13.4 (10.1, 16.7)	15.7 N/A**	13.8 N/A**	10.7 (7.2, 14.2)	13.9 (8.9, 18.9)	15.4 (4.1, 26.7)
Average # of Hospital Days per Recipient in final 6 months	20.0 (14.6, 25.4)	13.6 (9.4, 17.8)	19.5 (14.8, 24.2)	13.6 (8.9, 18.3)	11.9 (8.5, 15.3)	12.2 (5.6, 18.8)	18.4 (5.0, 31.8)

95% confidence intervals appear in parentheses.

*In this tabulation "admissions" were counted as the number of different months during which a decedent was admitted. Since a decedent might have more than one admission in a single month, these counts slightly underestimate the true admission rate, and result reciprocally in slightly overestimating the mean length of stay.

**Data needed for the calculation of this confidence interval are not available for these years.

those years, suggesting the possibility of an infectious disease epidemic. There was no consistent trend in age-adjusted mortality rates for the much smaller noninstitutionalized caseload.

To determine whether these changes were specific to the institutionalized Medicaid elderly, we also examined age-adjusted mortality rate trends in the general population of approximately 110,000 persons age 65 and over (Medicaid and non-Medicaid) in Hennepin County. Figure 2 shows that the age-adjusted mortality rates for Hennepin County elderly

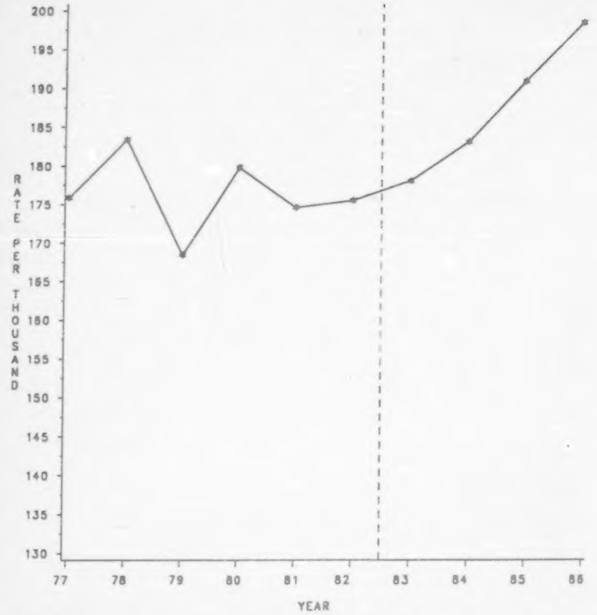

FIGURE 1—Age-Adjusted Mortality Rate, Institutionalized Medicaid Recipients, Age 65+

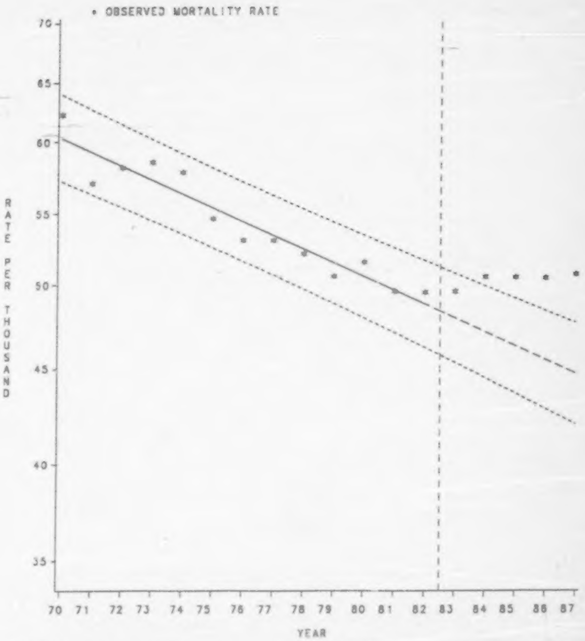

FIGURE 2—Age-Adjusted Mortality Rate for Estimated and Projected Hennepin County Population, Age 65+; Linear Regression and 95% Confidence Intervals for Years 1970–82 Projected for 1983–87

followed a roughly linear pattern of decline during the pre-cost containment period, falling from 62 per 1,000 in 1970 to 48/1,000 in 1982, a 22.5 percent reduction. Nationally, during a comparable 13-year period, 1968–80, there was a 24.2 percent reduction in age-adjusted mortality for persons age 65 and above.[13]

A linear regression line was fitted to the log-transformed 1970–82 observations to estimate the trend during the pre-cost containment period. Both the regression estimate and the 95 percent confidence interval for the pre-cost containment observations were projected through the year 1987 for comparison with observations in the post-cost containment period (1984–87). Figure 2 shows that, during 1984–87, the years following the institution of the PPS and other cost containment measures, the estimated age-adjusted mortality rates have ceased to fall and, in fact, are above the 95 percent confidence interval associated with the trend of the previous 13 years. While this does not represent an increase in the mortality rate from pre-cost containment years, it is a noteworthy departure from the mortality rate trend. It should be noted that the random variation in the mortality rate does not permit the identification of the specific year in which the mortality rate stops declining. We have selected 1983 as a break point because of the major policy changes instituted that year. Even if the break in the trend had actually begun in an earlier year, our findings of significant deviations in 1984–87 would still hold.

Discussion

These data include a large reduction in hospital admission rates and average length of stay by Hennepin County Medicaid recipients following the implementation of the PPS and other cost containment measures. The reduction in average hospital length of stay comes as no surprise. Data published in 1986 by the Health Care Financing Administration indicate that the average number of hospital days per Medicare admission to Minnesota decreased from 9.4 in 1982 to 7.3 in 1985—a 22.3 percent reduction.[14] The National Hospital Discharge Survey showed a 13.9 percent decline nationally in the average length of stay among patients age 65 and above from 1982 to 1985.[15] To a lesser extent, length of stay also declined for younger patients (11.4 percent for 45–64 year olds and 5.9 percent for 15–44 year olds). The large reduction in hospital admission rates among Medicaid recipients in our study differs from the experience in Wisconsin as reported by Sager, but is consistent with the National Hospital Discharge Survey data* and with impressions from the field that physicians have reduced their use of hospital care in response to pressure from third party payers. The reduction in days of hospital care per Hennepin County Medicaid recipient from 1982 to 1986 (53 percent for institutionalized and 55 percent for non-institutionalized recipients) far exceeds the amount one might estimate by looking only at changes in average length of stay.

The decrease in hospital care was not limited to the "healthier" members of the caseload. Recipients who died had comparable reductions in the first year following implementation of the PPS, although not in subsequent years.

Of more concern is the trend toward increasing age-adjusted mortality for the institutionalized elderly Medicaid recipients and the cessation of the downward trend in age-adjusted mortality for the general elderly Hennepin County population as a whole.

There are several possible explanations for these findings and we do not know which of them contributes most heavily. Reductions in hospital use—either decreased admission rates or shorter length of stay—may have contributed to halting the downward trend in mortality rates. Changes in quality of care, including premature or inappropriate hospital discharge to nursing homes of severely ill patients or reduced intensity of appropriate in-hospital services, may also explain these findings. Both of these explanations are consistent with some of the hypothesized but unproven effects of PPS.

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*The National Discharge Survey showed a decrease of 7.6 percent from 1982 to 1985 in discharges per 1,000 age 65 and older, from 428.1 to 393.2.[13]

An alternative or contributory explanation may be the changing nursing home case mix resulting not only from early hospital discharge under PPS but from the moratorium on new nursing home beds and the introduction of preadmission screening and expanded alternative care services. By keeping less severely ill patients out of nursing homes, these programs may have the effect of increasing the severity of illness, and thus the mortality rate, in the nursing home population. While this is a plausible explanation for the increasing mortality rate in the Medicaid nursing home caseload, it does not explain the leveling of the mortality rate trend in the general elderly Hennepin County population. In addition, increased mortality rates became apparent before nursing home utilization controls were fully implemented and before the Hennepin County Medicaid nursing home caseload stopped growing (it peaked in mid-1985). The moratorium on nursing home bed construction followed the largest expansion of nursing home beds in 10 years. Full implementation of preadmission screening was not reached until mid-1986.

Finally, there have been secular changes in attitudes about appropriate levels of aggressiveness in the care of the very old and of the terminally ill. Many physicians, patients and family members now favor less intensive or heroic treatment of such patients, which may result in lowering the mean age at death and in increasing death rates in nursing homes.

Several potential limitations must be considered in interpreting these data. First, the data are from one county (albeit a very large one) and may not be consistent with trends in other areas. A preliminary examination of statewide Medicaid data has, however, revealed similar trends in nursing home mortality rates throughout Minnesota. Second, the use of case closing codes to calculate mortality rates for Medicaid recipients may introduce some error, although Hennepin County Medicaid staff were confident that the reporting of deaths is complete and accurate for those in nursing homes and there was no indication that coding practices changed over time. Third, the finding of a change in the mortality trend for the general Hennepin County elderly population is dependent upon the accuracy of the Hennepin County population projections used in the analysis. If the population projections erred on the low side, they would yield an inflated age-adjusted mortality rate. While the accuracy of the projections cannot be verified until after the 1990 census, they are thought to be reliable for the relatively stable elderly population. The projections agreed closely with other Hennepin County population projections made independently by the State Demography Unit and by the State Health Department (personal communication).[16] The substitution of those projections in our analysis would not have altered our findings.

We cannot conclude that the change in the Hennepin County mortality rate trend is caused by the policy changes we have noted, but the data are troubling and underscore the need for analyses at multiple levels. Needed first is an examination of age-adjusted mortality trends at multiple sites, controlling for differences in population characteristics, Medicaid eligibility, utilization control policies, health care resources, and the prevalence of HMO enrollment. In addition, there is an urgent need to examine the access to care and the quality and appropriateness of care received by elderly Medicare and Medicaid patients, particularly those with life-threatening or terminal illnesses, and those in nursing homes.

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Prevalence of Depressive Symptoms in a Japanese Occupational Setting: A Preliminary Study

27

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Abstract: We measured the prevalence of depressive symptoms in 2,190 Japanese tax office workers using the Japanese version of the Center for Epidemiologic Studies Depression Scale (CES-D). Score distribution by sex was more symmetrical and the mean score of each sex was higher than in the United States population. A high level of depressive symptoms was found in 15.2 percent of males and 10.6 percent of females by controlling for age and marital status. Males aged 50 years and over had more depressive symptoms than other male age groups. Perceived stress, related both to family life and the workplace, was associated with a high level of depressive symptoms. "Long-distance marriage" ("business bachelorhood"), peculiar to Japanese occupations, had little influence on depressive symptomatology. (*Am J Public Health* 1989; 79:1486–1489.)

Introduction

In industrial countries, social and workplace environments have become more complicated and stressful. Recent epidemiologic studies in the United States and some European countries have found depressive disorders to be the most common among mental disorders in the community.[1] Advances in the psychiatric epidemiology of depression include the introduction of operational classificatory schemes[2] and the application of conventional self-rating measurements to population prevalence studies.[3–6] In Japan, there are few epidemiologic studies in these areas.

Self-rating depression scale, the Center for Epidemiologic Studies Depression Scale (CES-D),[7] has been widely applied to population surveys in the US and satisfactory levels of validity and reliability have been reported.[3–14] The CES-D was translated into Japanese by Shima, *et al*,[15] in 1985. In a comparison between depressed patients and normal controls, they found that the CES-D in Japanese translation had clinical validity and that its standard cut-off point of 15/16 was appropriate for Japanese use.[15] We also found the CES-D had a good level of concurrent validity with other indices of depression and psychological distress and an acceptable level of internal reliability in a sample of Japanese workers.[16,17]

The CES-D has also been applied in several surveys of other ethnic groups,[18–23] but it has been rarely used in an occupational setting, although some studies reported the values of depressive symptomatology based on the CES-D for a subsample of the employed in the surveyed community populations.[6,8,12,14,19,24,25] In this study we present basic data on the prevalence of depressive symptomatology as measured by the CES-D in a Japanese occupational setting.

Methods

Subjects

All 2,190 workers ages 19–63 years of 29 tax offices in Hokkaido, the northern part of Japan, were asked to complete the CES-D, other psychiatric scales, and several additional questions about sociodemographic status and psychosocial factors. The survey was carried out in September 1986. Complete CES-D data were obtained from 2,016 subjects (92.1 percent; 1,870 males, 142 females, and four with sex not specified).

The subjects were approximately the same educational level (years of education = 13), stable income-earners; all were full-time employees. Few of the subjects were separated or divorced. Generally they seemed to be representative of the workers of the Japanese government offices such as the tax office, even with respect to the low proportion of female workers.

Measurements

The Japanese translated version of the CES-D[15] has 20 items rated on a four-point Likert scale ("rarely or none of the time" = 0 to "most or all of the time" = 3). A score of 16 or higher indicates a high level of depressive symptoms as used both in the United States[7–10] and Japan.[15] Each psychosocial factor was measured by one item on a five-point rating scale with response categories (and scores) from "not at all (1)" to "a large amount (5)".

Analyses

The mean CES-D score and the proportion of subjects with high level of depressive symptoms value (CES-D score

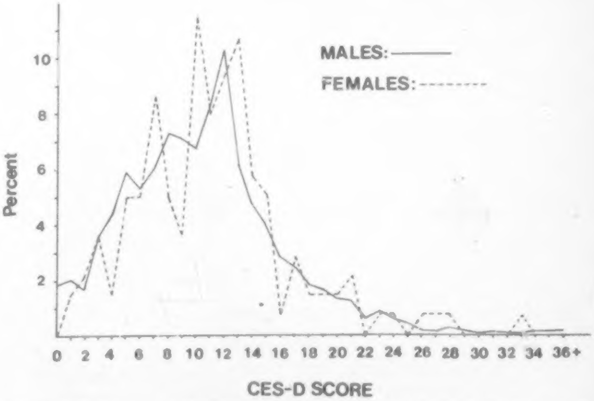

FIGURE 1—Percentage distribution of the Japanese version CES-D score by sex in a sample of Japanese workers.

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of 16 or higher) were calculated by sociodemographic status and psychosocial factors. The adjusted values, controlling for the effects of age (generation) and/or marital status, were also estimated. Because there were few separated or divorced subjects, this category was combined with the "never married". Analyses were performed separately by sex.

TABLE 1—Mean CES-D Score and Percentage of the Subjects with High Level of Depressive Symptoms (CES-D score of 16 or more) among Japanese Workers Ages 19–63 Years by Sociodemographic Status and Psychosocial Factors

	Males					Females				
		Mean CES-D score		% of High Scorers			Mean CES-D score		% of High Scores	
Factors	N	Crude	Adjusted	Crude	Adjusted	N	Crude	Adjusted	Crude	Adjusted
Total sample	1,793	10.5	10.5	15.0	15.2	132	11.1	10.5	14.1	10.6
	(77)[a]					(10)				
Sociodemographic Status										
Age (years)[b]										
19–29	414	11.2	10.2	18.8	12.2	56	11.6	11.1	21.4	18.3
30–39	633	9.9	10.1	11.9	13.3	20	12.1	12.5	15.0	17.2
40–49	267	10.3	10.7	12.7	15.4	25	9.8	10.1	0.0	2.1
50–63	479	10.7	11.1*	16.9	19.3*	21	10.5	10.9	12.5	15.5
	(77)					(10)				
Marital status[c]										
Married	1,423	10.1	10.0	12.7	12.1	64	10.0	10.0	6.3	7.6
Never married or separated/divorced	370	11.9	12.2*	23.8	26.0***	68	12.1	12.1*	22.1	20.8*
	(77)					(10)				
Number of children[d]										
0	163	10.1	10.3	14.7	16.4	14	10.1	9.9	7.1	3.3
1	315	10.5	10.6	14.9	15.6	14	8.6	8.6	7.1	5.7
2	804	9.9	9.8	11.6	11.1	31	10.6	10.7	6.5	8.3
3+	141	10.3	10.2	11.4	10.8	5	9.6	9.7	0.0	3.2
Residential status[c]										
Living with family	1,373	10.2	10.2	13.2	13.4	102	10.8	10.9	13.7	14.1
Long-distance marriage	154	10.8	10.6	16.2	14.4	3	5.7	4.5	0.0	0.0
Living alone	254	11.7	11.6**	23.2	23.3***	24	12.4	12.3	20.8	19.8
	(89)					(13)				
Psychosocial factors										
Conjugal Relationship[d]										
Very good (1)	305	8.0	8.0	4.6	4.8	14	8.5	8.3	0.0	0.0
2	505	10.0	10.0	12.5	12.6	15	9.0	8.8	0.0	0.0
3	594	11.0	11.0	15.8	15.6	28	11.2	11.4	14.3	15.0
Bad/very bad (4/5)	10	23.1	23.3***	70.0	70.9***	—	—	—	—	—
	(9)					(7)				
Parental relationship[d]										
Very good (1)	309	8.2	8.2	5.5	5.7	13	8.5	8.1	0.0	0.0
2	491	9.9	10.0	12.0	12.2	20	9.7	9.9	0.0	1.7
3	517	11.2	11.2	16.3	16.0	22	11.6	11.7	18.2	18.1*
Bad/very bad (4/5)	10	15.1	15.0***	20.0	19.6***	—	—	—	—	—
	(96)					(9)				
Perceived stress related to family life										
Not at all (1)	675	8.6	8.6	6.4	6.0	35	10.3	9.5	8.6	1.9
2	860	10.8	10.8	15.7	15.9	70	11.0	11.3	14.3	17.2
3	185	13.7	13.8	32.4	32.9	21	10.6	11.0	14.3	16.4
A large amount (4/5)	49	18.1	17.9***	55.1	53.8***	5	16.4	16.0	40.0	37.4
	(101)					(11)				
Perceived stress related to the workplace										
Not at all (1)	286	7.6	7.5	2.8	2.2	18	7.0	6.6	0.0	0.0
2	867	9.4	9.5	9.1	9.2	62	10.4	10.6	6.4	6.1
3	494	12.4	12.5	24.3	24.6	35	11.0	10.9	14.3	14.0
A large amount (4/5)	121	16.6	16.5***	47.9	47.8***	16	17.7	17.3***	56.3	54.5***
	(102)					(11)				
Seeking medical counseling										
Yes (wanted)	216	12.0	12.1	26.4	26.3	6	18.7	18.6	50.0	49.6
No (not wanted)	1,306	10.1	10.2***	13.6	13.7***	99	10.0	10.0***	11.1	11.1***
	(348)					(37)				

Note: "adjusted" values are estimated by controlling for age (generation) and marital status.
N: Number of the subjects in the subcategory.
a) Number of missing subjects is shown in brackets.
b) The values adjusted with controlling the effect of marital status are presented.
c) The age-adjusted values are presented.
d) Only married subjects were analyzed, so that the age-adjusted values are presented.
*, **, and ***: significance level of $p < 0.05$, $p < 0.005$, and $p < 0.001$, respectively.

TABLE 2—Selected Results of Reported Mean CES-D Score and Percentage of High Level of Depressive Symptoms (CES-D score of 16 or more) for US and Mexican Workers Based on Community Surveys[a]

Study	Research Site Sample of Workers	Value of Depressive Symptomatology (adjusted value) Mean	Rate
Comstock & Helsing[8]	Washington County		%
(1976)	Whites	—	14.4 (15.1)
	Blacks	—	22.4 (12.3)
	Kansas City Whites	—	17.1 (17.8)
Eaton & Kessler[12]			
(1981)	HANES sample	—	13.1 (15.7)[b]
Frerichs, *et al.*[25]			
(1981)	Los Angeles county	8.0	14.0 (13.7)
Aneshensel, *et al.*[24]	Los Angeles county		
(1981)	Males	4.32 (4.35)	—
	Married/Unmarried	3.76/5.52 (4.14/5.26)	
	Females	5.57 (5.15)	—
	Married/Unmarried	5.17/8.23 (5.57/5.62)	
Gary[14]			
(1985)	A northeastern city		
	Selected Black males	10.78	—
Vega, *et al.*[19]	Tjuana, Mexico	8.07	—
(1987)	Mexican		
Mendes de Leon &	San Antonio, Texas		
Markides[8]	Mexican-Americans		
(1988)	Middle-aged	7.4 (7.5)	11.0 (12.0)
	Younger-aged	8.1 (8.6)	12.0 (15.0)
Present study	Hokkaido, Japan		
	Tax workers		
	Males	10.5 (10.5)	15.0 (15.2)
	Females	11.1 (10.5)	14.1 (10.6)

Adjusted values are generally estimated by controlling for sex, age, marital status, education, and income.

a) Only the results of full-time employees are presented.

b) The effect of race was also taken into account.

Results

Figure 1 displays the percentage distribution of the CES-D scores by sex. Although the distribution of females had dichotomous peaks (scores at 10 and 13) and was not artificially smoothed, there was a much smaller number of female subjects, so that the distribution of each sex was relatively similar and not heavily skewed (males: mode = 12, skewness = 1.04; females: mode = 10, skewness = 0.94) therefore standard parametric tests on these data were deemed to be appropriate.

Table 1 shows the mean CES-D score and the proportion of subjects with high level of depressive symptoms by sociodemographic status and psychosocial factors. There were no sex differences in either indicator of depressive symptomatology. When adjusted for the effect of marital status, a weak linear relationship with age was found in males. Males ages 50 years or more had more depressive symptoms than other age groups. For other sociodemographic status, both male and female subjects who were unmarried (never married or separated/divorced) and living alone had a higher level of depressive symptoms.

Among psychosocial factors, the subjects who perceived poor relationships with spouse and with parents were more depressed as were subjects who reported more stress in their family life and in the workplace. Subjects who desired medical counseling were also more depressed.

Discussion

United States studies have reported values of depressive symptomatology based on the CES-D for the employed in community populations as displayed in Table 2. Since the present results were derived from an occupational survey, they may not be comparable to the values in the table. Mean CES-D scores for both sexes were higher in our study than in the US employed, except for the Black men surveyed by Gary[14]; nevertheless the percentages of high level depressive symptoms were similar to the US employed.

One reason for the disparity of mean score between Japan and the United States may be the difference in response frequencies of the negative and positive items. The Japanese tend to choose an intermediate response to the positive items when compared to the US population so that the score distributions of the scale of Japanese subjects show generally lower frequencies than the US counterparts at the low symptom portion. Such a tendency may reflect the sociocutural and/or personality characteristics of Japanese who tend to avoid extreme answers to anything.

In Japan, many male employees have long-distance marriages (business bachelorhood, living alone separated from family members as a result of being promoted to a new job in a distant place). This situation may last for several years. Males in the subclass of the long-distance marriages were somewhat more likely to have depressive symptoms than those who were living with family members, but far less than others who were living alone.

Kessler and Essex[26] and Gove, *et al*,[27] reported that marriage had positive effects on depression and psychological well-being. The present study supports this observation; it also demonstrates that perceived stress in family life and in the workplace is related to depressive symptomatology. Worker depression has been related to such factors as work overload and job pressures in other studies.[28,29]

The limitations of the present results include the nature of the sample and the fact that each psychosocial factor is measured by a single question; a direct question about self-perceived job stress has been reported as a good predictor of subsequent ill health.[29] This should be considered a preliminary study of the mental health of Japanese workers. Further research is needed based on several samples of Japanese workers and a more thorough exploration of stress to examine the factors affecting depressive symptomatology of Japanese workers.

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For further information, contact Ellen Lemberg, RN, MS, Box C288, Continuing Education/Extended Studies, School of Nursing, University of Colorado Health Sciences Center, 4200 East 9th Avenue, Denver, CO 80262; (303) 270-8691.

Person-Years of Life Lost Due to Cancer in the United States, 1970 and 1984

JOHN W. HORM, MSC, AND EDWARD J. SONDIK, PHD

Abstract: The number of deaths due to cancer in the United States reached an all-time high of 453,450 deaths in 1984 and, due to the dynamics of population growth, will continue to increase if the risk of dying from cancer does not change.

Between 1970 and 1984, the total Person-Years of Life Lost (PYLL), the sum of the difference between the actual age at death and the expected remaining lifetime for each person who died of cancer, increased for most cancer sites as well as for all sites combined. In 1984, 6,881,281 person-years of life were lost due to cancer deaths, up from 5,303,668 in 1970. The exceptions are those cancers for which there has been major progress in either prevention or treatment; e.g., stomach and cervix uteri (prevention) and testicular, Hodgkin's disease, leukemia, and childhood cancers (treatment).

The Average Years of Life Lost (AYLL) per person dying from cancer in 1984 was generally less than in 1970. Overall, each person who died from cancer in 1984 died 15.2 years earlier than his/her life expectancy. The greatest loss was for those who died of childhood cancers (66.9 years earlier), followed by testicular cancer (35.8 years earlier). The least loss relative to the expectation of life was for those who died of prostate cancer. The 25,400 men who died from prostate cancer in 1984 died an average of nine years earlier than otherwise expected. (*Am J Public Health* 1989; 79:1490–1493.)

Introduction

Deaths due to cancer accounted for 453,450 deaths in 1984[1] in the United States, second only to heart disease. Many of these deaths may be postponed or avoided entirely through the use of appropriate interventions such as primary prevention, screening/early detection, and increased application of state-of-the-art treatment.[2] A death which occurs before the end of average life expectancy is, by definition, a premature death. The age-adjusted and age-specific cancer mortality rates do not fully address the issue of premature mortality and therefore other measures are needed.[3]

Person-Years of Life Lost (PYLL) considers a cancer death to be premature in that the death occurred earlier than it would have occurred in the absence of that cancer.

Average Years of Life Lost (AYLL) is the average of the differences between the actual ages at death and the expected remaining years of life for each person who died of cancer.

In our computation of PYLL and AYLL we have used age-based and sex-specific estimates of the length of life remaining rather than fix an upper bound on length of life at age 65 such as has been used by others.

Taken together, the PYLL and the AYLL reflect the cost of dying from cancer in terms of lost years of life expectancy. They may also be used to assist in the evaluation of cancer control progress and to provide guidance for resource allocation in cancer prevention and control. We examined PYLL and AYLL for cancer in 1970 and 1984.

Methods

The numbers of cancer deaths for both 1970 and 1984 and information on the underlying cause of death as well as demographic information such as age, sex, race, and other items for each death occurring annually in the United States were obtained from the National Center for Health Statistics (NCHS). The deaths have been categorized by anatomic site of the cancer, sex, and age at death for each calendar year.

For each cancer death which occurred in either 1970 or 1984, the PYLL was estimated by taking the difference between the actual age at which the person died and the expectation of remaining life for an individual matched by age and sex for each of 1970 and 1984 as obtained from life tables published yearly by the NCHS.[5,6] These differences were then summed over all persons dying of cancer for 1970 and for 1984.

The AYLL is a statistical average arrived at by simply dividing the total PYLL by the number of people affected. From Table 1, 4,564 women died prematurely from cancer of the cervix uteri in 1984 for a total of 105,899 person-years of life lost or an average of 23.2 years of life lost for each of the 4,564 women.

Results

Tables 1, 2, and 3 have for both 1970 and 1984 the numbers of cancer deaths in the United States, the total number of person-years of life lost due to cancer, and the average years of life lost for those who died of cancer. The cost in average years of life lost was greater for women (17.1) than for men (13.5) in 1984, due in part to the greater life expectancy for women compared to men, 78.2 years and 71.2 years, respectively. Even though there were fewer women than men dying from cancer in 1984 (210,687 versus 242,763), the women lost more potential years of life, 3.6 million compared to 3.3 million for men. For both sexes combined, lung cancer caused the loss of the most total person-years of life in 1984 followed by breast and colorectal cancer (Figure 1). Deaths due to testicular cancer, because of the small numbers of deaths, cost the fewest total person-years of life among those cancer sites investigated.

In contrast, the greatest average years of life lost per person dying of cancer was for childhood cancers (Figure 2), followed by deaths due to testicular cancer, Hodgkin's disease, cancer of the cervix uteri, brain cancer and finally malignant melanoma, each of which caused death more than 20 years before the life expectancy.

In 1984 the most costly cancer for women was cancer of the breast which caused a loss of over 760,000 person-years of life among 39,470 women, up 28 percent from 1970. These women died an average of 19.3 years earlier than expected. Mass screening programs have suggested that the mortality

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TABLE 1—Potential Years of Life Lost (PYLL) and Average Years of Life Lost (AYLL) due to Premature Deaths from Cancer in the United States, all Races, both Sexes, 1970 and 1984

Cancer Sites	Number of Deaths		PYLL		AYLL	
	1970	1984	1970	1984	1970	1984
All sites	329,375	453,450	5,303,668	6,881,281	15.1	15.2
Oral cavity	7,610	8,461	117,854	134,095	15.5	15.8
Esophagus	5,969	8,657	87,905	126,758	14.7	14.6
Stomach	16,020	14,246	209,706	191,045	13.1	13.4
Colon & rectum	45,924	57,510	615,264	744,941	13.4	13.0
Pancreas	17,885	23,154	246,952	315,020	13.8	13.6
Lung	65,003	118,565	989,335	1,757,001	15.2	14.8
Melanoma (skin)	3,146	5,377	66,900	110,765	21.3	20.6
Breast	29,912	39,720	597,050	763,511	20.0	19.2
Cervix uteri	6,547	4,564	146,261	105,899	22.3	23.2
Corpus uteri	5,468	5,965	88,100	89,563	16.1	15.0
Ovary	9,831	11,208	193,462	200,279	19.7	17.9
Prostate gland	17,249	25,400	156,281	228,597	9.1	9.0
Testis	760	401	26,693	14,343	35.1	35.8
Urinary bladder	9,011	9,714	101,023	103,176	11.2	10.6
Kidney	6,103	8,411	100,310	127,937	16.4	15.2
Brain & CNS	6,027	9,866	153,882	215,805	25.5	21.9
Hodgkin's disease	3,410	1,960	89,249	53,022	26.2	27.1
Non-Hodgkin's lymphoma	9,592	14,486	176,232	232,071	18.4	16.0
Multiple myeloma	4,417	7,470	63,042	100,736	14.3	13.5
Leukemia	14,492	17,212	333,439	320,480	23.0	18.6
Childhood	3,611	1,884	233,435	125,951	64.6	66.9

TABLE 2—Potential Years of Life Lost (PYLL) and Average Years of Life Lost (AYLL) due to Premature Deaths from Cancer in the United States, all Races, Males, 1970 and 1984

Cancer Sites	Number of Deaths		PYLL		AYLL	
	1970	1984	1970	1984	1970	1984
All sites	179,352	242,763	2,558,996	3,284,558	14.0	13.5
Oral cavity	5,495	5,764	79,810	87,527	14.5	15.2
Esophagus	4,442	6,312	62,308	90,091	14.0	14.3
Stomach	9,802	8,469	121,490	107,681	12.4	12.7
Colon & rectum	22,117	27,989	269,512	338,649	12.2	12.1
Pancreas	10,061	11,516	130,937	149,202	13.0	13.0
Lung	52,680	82,385	751,081	1,113,112	14.3	13.5
Melanoma (skin)	1,735	3,207	34,394	62,209	19.8	19.4
Breast	265	250	3,552	3,409	13.4	13.6
Prostate gland	17,249	25,400	156,281	228,597	9.1	9.0
Testis	760	401	26,693	14,343	35.1	35.8
Urinary bladder	6,257	6,600	66,853	66,762	15.3	14.4
Kidney	3,858	5,201	59,091	74,664	15.3	14.4
Brain & CNS	3,434	5,395	81,686	111,329	23.8	20.6
Hodgkin's disease	2,016	1,204	50,566	31,461	25.1	26.1
Non-Hodgkin's lymphoma	5,233	7,312	94,256	115,937	18.0	15.9
Multiple myeloma	2,340	3,737	30,269	46,449	12.9	12.4
Leukemia	8,128	9,378	174,139	166,464	21.4	17.8
Childhood	2,039	1,073	125,194	68,257	61.4	63.6

from breast cancer can be reduced by 30 percent through screening.[7,8] If so, a screening program could prevent about 11,800 breast cancer deaths per year. If these 11,800 women were to live to their full life expectancy, about 227,000 person-years of life could be saved yearly.

The 36,180 women who died from lung cancer in 1984 lost a total of 643,889 person-years of life, almost triple the 1970 figure of 238,254 person-years. In 1984, these women died an average of 17.8 years before expected, an improvement over 1970 when female lung cancer victims died 19.3 years before their life expectancy. It is believed that about 75 percent of lung cancer deaths among women are caused by cigarette smoking.[9] Preventing 75 percent of these lung cancer deaths could result in the postponement of death by some 19 years for over 27,000 women annually. This would result in the saving of over 500,000 person-years of life every year.

Additionally, 75 percent of the deaths due to cancer of the cervix uteri are thought to be preventable through screening with the Pap smear.[10] Pap smear screening could potentially extend the lives of 3,400 women by 23 years each for a total savings of 78,200 person-years of life.

Dramatic improvements have been noted in the person-years of life lost among women between 1970 and 1984 for Hodgkin's disease with a 44 percent decrease and, most dramatically, childhood cancers with less than half as many person-years of life lost in 1984 than in 1970. Smaller decreases are seen for cancers of the stomach and for

TABLE 3—Potential Years of Life Lost (PYLL) and Average Years of Life Lost (AYLL) due to Premature Deaths from Cancer in the United States, all Races, Females, 1970 and 1984

Cancer Sites	Number of Deaths		PYLL		AYLL	
	1970	1984	1970	1984	1970	1984
All sites	150,023	210,687	2,744,672	3,596,723	18.3	17.1
Oral cavity	2,115	2,697	38,043	46,568	18.0	17.3
Esophagus	1,527	2,345	25,597	36,667	16.8	15.6
Stomach	6,218	5,777	88,216	83,364	14.2	14.4
Colon & rectum	23,807	29,521	345,751	406,293	14.5	13.8
Pancreas	7,824	11,638	116,015	165,817	14.8	14.2
Lung	12,323	36,180	238,254	643,889	19.3	17.8
Melanoma (skin)	1,411	2,170	32,507	48,556	23.0	22.4
Breast	29,647	39,470	593,498	760,102	20.0	19.3
Cervix uteri	6,547	4,564	146,261	105,899	22.3	23.2
Corpus uteri	5,468	5,965	88,100	89,563	16.1	15.0
Ovary	9,831	11,208	193,462	200,279	19.7	17.9
Urinary bladder	2,754	3,114	34,171	36,414	12.4	11.7
Kidney	2,245	3,210	41,219	53,273	18.4	16.6
Brain & CNS	2,593	4,471	72,197	104,476	27.8	23.4
Hodgkin's disease	1,394	756	38,683	21,561	27.7	28.5
Non-Hodgkin's lymphoma	4,359	7,174	81,976	116,135	18.8	16.2
Multiple myeloma	2,077	3,733	32,773	54,287	15.8	14.5
Leukemia	6,364	7,837	159,300	154,015	25.0	19.7
Childhood	1,572	811	108,241	57,695	68.9	71.1

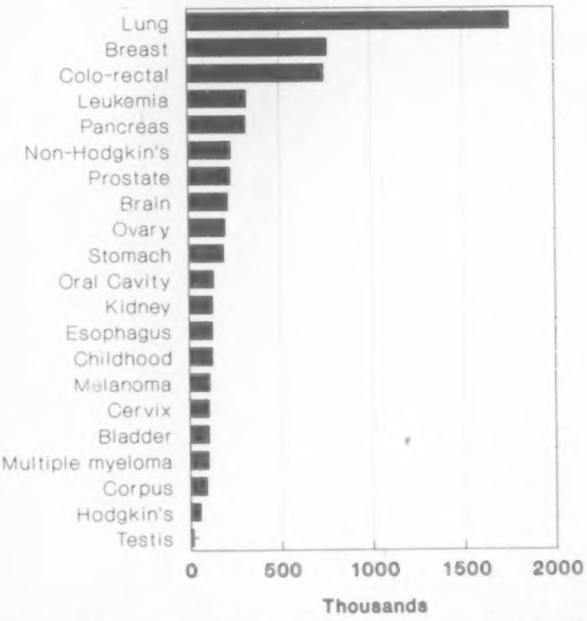

FIGURE 1—Person-Years of Life Lost (PYLL) due to Cancer, all Races, both Sexes, 1984

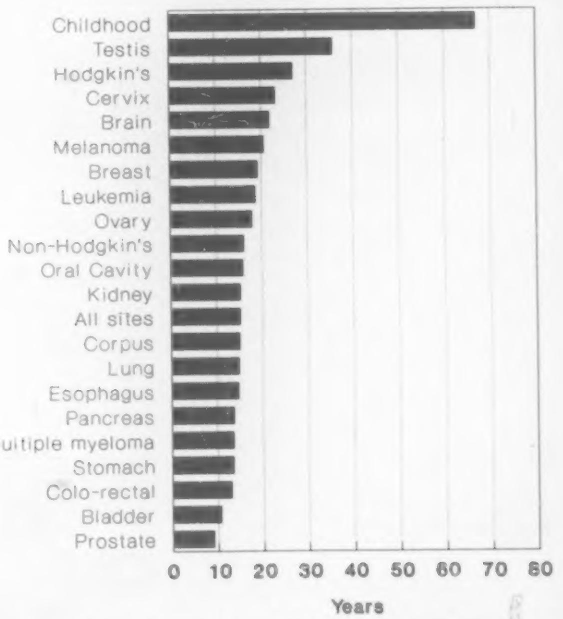

FIGURE 2—Average Years of Life Lost (AYLL) due to Cancer Deaths, all Races, both Sexes, 1984

leukemias.

Among men, lung cancer was the most deadly cancer causing the loss of the most person-years of life in 1984. Over 1.1 million person-years of life were lost among 82,385 men who died of lung cancer an average of 13.5 years earlier than otherwise expected. If 80 percent of these deaths could be avoided through prevention, almost 900,000 person-years of life could be gained annually.

Colorectal cancer deaths accounted for 338,649 lost person-years of life in 1984, up by 26 percent since 1970. Each man who died from colorectal cancer in 1984 died, on the average, 12.1 years before he would have otherwise expected to die.

Deaths from prostatic cancer among 25,400 men cost almost 229,000 person-years of lost life in 1984 as these men died nine years before their average life expectancy would predict.

Five cancer sites among those examined actually demonstrated decreases in the number of person-years of life lost due to cancer deaths between 1970 and 1984. Both testicular and childhood cancer had decreases in the person-years of life lost of about 45 percent from 1970 to 1984 amounting to a savings of 69 thousand person-years of life in 1984 alone. The improvement in testicular cancer is primarily due to the development of curative chemotherapy for nonseminoma cases which has increased the five-year survival rate to over 80 percent.[14,15] Although testicular cancer is relatively rare, it causes death an average of 35.8 years before the normal life expectancy. Male childhood cancer victims died 63.6 years earlier than expected in 1984 with 68,257 person-years of life lost among 1,073 boys. Additional savings are seen for Hodgkin's disease, a 38 percent improvement; stomach cancer, an 11 percent improvement; and a 4 percent improvement for leukemia.

Discussion

The methodology used here differs from that used in previous investigations in that here we have used the life expectancy as the endpoint rather than age 65.[11–13] We believe that the life expectancy is a more appropriate endpoint for such an analysis because a large proportion of cancer deaths, about 65 percent, as well as 70 percent of non-cancer deaths occur over the age of 65 and today people remain productive and active well beyond the classical retirement age of 65. Further, we would argue that any death which is either preventable or avoidable, and which occurs before the end of the affected person's normal lifespan, is premature and hence should contribute to person-years of life lost. It should be noted that in 1984, a person 65 years old could expect to live for another 16.8 years.

The PYLL and AYLL are estimates of the differences between the actual age at death and the expected age at death considering all possible causes of death weighted by their occurrence in the population at large. These estimates do not depend on the date of diagnosis nor on the time between diagnosis and death (survival) and thus are free of some of the problems inherent in the interpretation of trends in incidence and survival rates due to diagnostic artifacts such as lead time bias.

Ideally we would like the PYLL to decrease over time. However, due to the aging of the population and to the increasing size of the population and therefore the greater numbers of persons at risk of death from cancer, it is possible for the PYLL to increase without an increase in the age-sex-specific risk of dying of cancer. The PYLL then estimates the actual impact of the cancer deaths on the population in terms of total person-years of life lost every year. As the impact of competing causes of death on the population lessens, the PYLL due to cancer will continue to become numerically larger in the United States for the reasons outlined above.

On the other hand, the AYLL (Average Years of Life Lost) estimates the joint effects of the changes in the cancer patients' ages at death and the change in the population life expectancy. The life expectancy at birth of the United States population increased by almost four years between 1970 and 1984, from 70.9 to 74.7, respectively. The expectation of remaining life also increased between 1970 and 1984 for all races combined, for each sex, and at every age with larger increases for younger persons and lesser increases for older persons. For making comparisons over time, we can artificially hold the overall life expectancy constant. In so doing, the comparison of two AYLLs shows the change in the average loss of expected life-years per cancer death.

It is important to note that the AYLL is based only on those people who die from cancer and therefore does not provide an estimate of either cure or prevention. For example, the number of people dying from testicular cancer decreased from 760 in 1970 to 401 to 1984 due to the advent of curative treatment but the AYLL increased because of different age and stage distributions between those who died and those who were cured. We also note that with no change in cancer patient survival, the AYLL would be expected to increase in accordance with the increasing life expectancy of the United States population. Therefore, we consider any changes in AYLL as a conservative (pessimistic) estimate of the curative life extension impact of cancer treatment.

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Breast Cancer Risk and Participation in Mammographic Screening

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Abstract: Within the context of an organized breast cancer screening program we conducted a prospective evaluation of the relation between breast cancer risk and participation in mammographic screening. The influence on participation of known breast cancer risk factors, as well as a summary risk label, (i.e. "high", or "moderate") were examined. The overall participation rate was 71 percent among 2,422 women, 50 to 79 years of age, invited to a centralized clinic. Multivariate analyses showed participation to be somewhat decreased among women with late menopause and definitely increased among women with any of the following factors: 1) increased age; 2) a family history of breast cancer; and 3) a previous breast biopsy. Women in the high-risk group were most likely to participate but the effect of the label was stronger among women ages 50 to 59 compared to women ages 60 to 79. The study results are generally consistent with previous findings that participants in screening programs have higher rates of breast cancer. The results also suggest the possibility that providing breast cancer risk information may encourage participation in screening. (*Am J Public Health* 1989; 79:1494–1498.)

Introduction

Self-selection bias has been a major concern in the assessment of cancer screening modalities.[1-3] The Health Insurance Plan (HIP) randomized trial of mammography demonstrated that breast cancer rates were higher among women who complied with an invitation to obtain a mammogram (participants) compared to those in the study population who did not comply with that invitation.[4] Several investigators have shown a higher prevalence of breast cancer symptoms among participants, but the influence of breast cancer risk factors on screening behavior has not been thoroughly evaluated.[5]

Investigators have examined the relationship between women's use of mammography and three risk factors: age, family history, and previous benign breast disease.[5-10] All but one of the studies were conducted retrospectively.[7] Participation appears to decrease with advancing age,[5-7] although one report showed increased participation,[8] and yet another found no association.[9] Two studies found no association between family history and participation.[5,6] A third study reported that participants in screening were more likely to have a family history of breast cancer, but this finding was based on very small numbers.[9] One study has examined the role of previous benign breast disease and showed an association in a negative direction with subsequent participation in screening, but the definition of this disorder was not explicit.[9]

The study reported here prospectively evaluates the association between participation in an organized screening program and the following six risk factors: age, family history of breast cancer, having a previous benign breast biopsy, nulliparity to age 30, early age at menarche, and late age at menopause. Previous work has suggested that perceived vulnerability to breast cancer is positively associated with participation in screening.[5,7,8] We, therefore, hypothesized that those factors which might be readily identified by a woman as increasing her risk of breast cancer (i.e., increased age, family history, and having needed a breast biopsy) would show positive associations with participation in screening. In contrast, we expected that nulliparity, early age at menarche, and late age at menopause would not be associated with participation in screening, as women are less likely to be aware that these factors increase their risk for breast cancer. In addition to the association between specific risk factors and participation in screening, this study also assessed the effect of being informed that one is at high risk vs moderate risk for breast cancer.

Methods

The study was conducted at Group Health Cooperative of Puget Sound (GHC), a 360,000-member closed panel health maintenance organization (HMO) that includes two hospitals and 21 primary care facilities in Western Washington state. Women GHC enrollees are slightly better educated and more likely to be in households with higher incomes than the general population of comparably aged women residing in Washington state.[11]

In March of 1985, Group Health Cooperative initiated a risk-based Breast Cancer Screening Program.[12,13] All female enrollees 40 years of age and older were mailed a two-page questionnaire which elicits information concerning breast cancer risk factors, medical and screening history, and selected lifestyle behaviors. Seven of 21 items on the questionnaire pertain to specific breast cancer risk factors. The response rate to this survey has been 85 percent.[13,14] Based on their personal constellation of risk factors, all women are assigned by the screening program to one of four risk categories: "high," "moderate," "borderline," or "no increased risk" which represent 18, 27, 12, and 43 percent of the population, respectively (see Appendix). Each woman who completes and returns this questionnaire is sent a follow-up letter, within two months, which indicates her risk category and a recommendation to: perform monthly breast self-examination, obtain a breast physical examination annually, and be seen in the breast cancer screening center for a mammogram every 1, 3, or 5 years according to risk level. Letters of invitation do not convey any risk factor information other than a woman's risk category. An automated data system maintains the survey data and documents all screening activity. These data served as the basis for the analysis of participation presented here.

The sample for this analysis consisted of women who had completed a risk factor questionnaire, were invited for a screening visit between March 1, 1985 and December 31, 1985, reported no mammogram during the one year prior to

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being surveyed, and were continuously enrolled at GHC since being invited to come in for screening. The study group was limited to women ages 50 to 79 who were in the "high" and "moderate" risk categories (Table 1). Of the 2,722 women in this group, 2,422 received invitations to the screening program during this period and constitute the study population. The non-study population consists of the 300 women from this group who did not receive invitations during the study period plus 4,498 women whose only risk factor was age. At the time of this study, these women were placed in a group designated as "no increased risk" and were not invited for a screening visit. This policy has subsequently changed.[13]

Participation was defined as scheduling and completing an appointment in the screening center by June 30, 1986. There was no fee for this visit which included instruction in breast self-examination and a physical examination of the breast, as well as the mammogram. Subjects had a minimum of six and maximum of 15 months opportunity to come in for screening following their invitation. At the time of this study, the wait time to obtain a mammogram was approximately one month. Analysis of overall program data shows that 93 percent of women who participate within one year do so within the first six months after being invited. Women received only one invitation and were not sent any reminders.

The relation between participation in screening and the following six factors was considered: age, family history of breast cancer, previous benign breast disease, menarche at 10 years of age or younger, menopause at 55 years of age or older, and nulliparity to age 30. A positive family history was defined as having one or more first or second degree family members with a history of breast cancer (i.e., mother, sister, daughter, grandmother, or aunt). Benign breast disease was defined as having reported one or more breast biopsies which were non-cancerous. In addition to considering the six individual risk factors, the effect on participation of being assigned a "high" vs "moderate" risk label was examined.

TABLE 1—Risk Factor Comparison in Study vs Non-Study Women

Characteristics	Study[a] Women (n = 2,422)		Non-Study[a] Women (n = 4,798)	
	Number	Percent	Number	Percent
Age (years)				
60–79	1515	62.6	2715	56.6
50–59	907	37.4	2083	43.4
Risk Category[b]				
High	875	36.1	193	4.0
Moderate	1547	63.9	107	2.2
No Increased Risk[c]	0	0	4498	93.8
Family History Breast Cancer				
Yes	886	36.6	499	10.4
No	1536	63.4	4299	89.6
Previous Benign Breast Biopsy				
Yes	541	22.7	383	8.2
No	1841	77.3	4281	91.8
Nulliparity to Age 30				
Yes	1856	35.3	163	3.4
No	1566	64.7	4634	96.6
Menarche Age < 10				
Yes	166	7.0	41	0.9
No	2214	93.0	4661	99.1
Menopause Age ≥ 55				
Yes	427	17.6	128	3.2
No	1995	82.4	3822	96.8

[a] Among GHC women 50 years of age and older who completed and returned a risk factor questionnaire before December 31, 1985 and had not reported having a mammogram within one year: "Study Women" are those women who were invited for screening; "Non-Study Women" represent those who were not invited by December 31, 1985.

[b] There are no "Borderline" risk women in the age group studied (see Appendix).

[c] These women have none of the risk factors listed, except age.

Bivariate contingency table analyses were carried out initially to assess the association between participation and each of the seven variables considered individually. Logistic regression was then used to estimate the effect of each variable controlling for the influence of all others. The logistic analysis was done in two steps. First, models were fit to test if there were any significant interactions between risk label and the other variables. Then, a final logistic model was fit containing the seven main effects plus any significant interactions.

Results

Since the study population was composed of women who were invited for screening because they were at increased risk for breast cancer, each woman necessarily had one or more risk factors for the disease. As expected, a higher proportion of study women had any given risk factor compared to non-study women (Table 1).

Table 2 shows the percentage of study women with each of the six risk factors according to their risk category. As expected, a larger proportion of high-risk women have any particular risk factor.

Of the 2,422 women in the study population, 71 percent came in to the screening center as requested by the letter of invitation. Table 3 shows the crude odds ratios (OR) for the association between breast cancer risk factors and participation in mammography. The strongest association is between the risk category and participation (OR = 2.59), followed by family history (OR = 1.83), previous biopsy (OR = 1.60), and age (OR = 1.45). Age is dichotomized in this bivariate analysis since participation varies little among women above age 60 when grouped into five-year intervals (73 percent to 75 percent). Three factors (age ≥ 55 at menopause, nulliparity to age 30, and age ≤ 10 at menarche) show no association with participation.

Table 4 shows odds ratios estimated from the logistic regression analysis. Since there is a significant interaction between age and risk designation, the final logistic model consists of the six risk factors, the risk designation, and this interaction term. Family history of breast cancer and previous benign breast biopsy continue to be associated with increased participation although the odds ratio is slightly less. Neither nulliparity to age 30 nor menarche at age 10 or

TABLE 2—Proportion of Women in Moderate and High-Risk Categories Who Have a Particular Risk Factor

Risk Factors	Risk Category[a]	
	Moderate (N = 1547)	High (N = 875)
Age 60–79 years	61%[b]	66%
Family History of Breast Cancer[c]	21%	63%
Previous Benign Breast Biopsy	15%	37%
Nulliparity to Age 30	31%	43%
Age ≤ 10 at Menarche	5%	10%
Age ≥ 55 at Menopause	14%	24%

[a] Risk category is the designation given by the screening program according to the algorithm in the Appendix.

[b] For example, 61 percent of moderate-risk women are 60 to 79 years of age compared to 66 percent of high-risk women.

[c] First and second degree family history result in different risk designations (see Appendix).

TABLE 3—Bivariate Analysis of Breast Cancer Risk and Participation in Mammographic Screening

Characteristics	% Participation	Unadjusted Odds Ratio (95% CI)[a]
Age (years)		
60–79	73.3	1.45
50–59	65.7	(1.21, 1.73)
Risk Category		
High	82.2	2.59
Moderate	64.1	(2.12, 3.15)
Family History of Breast Cancer		
Yes	78.2	1.83
No	66.2	(1.52, 2.22)
Previous Benign Breast Biopsy		
Yes	77.6	1.60
No	68.4	(1.28, 2.00)
Nulliparity to Age 30		
Yes	71.3	1.05
No	70.2	(.87, 1.26)
Age ≤ 10 at Menarche		
Yes	68.7	.90
No	70.7	(.64, 1.26)
Age ≥ 55 at Menopause		
Yes	66.4	.84
No	71.4	(.67, 1.05)

[a] 95% confidence interval for odds ratio.

[b] Referent group for odds ratio is women without the risk factor.

TABLE 4—Results of Logistic Regression Analysis of Breast Cancer Risk and Participation in Mammographic Screening

Characteristics	Adjusted Odds Ratio[a]	(95% CI)
Family History Breast Cancer	1.35	(1.02, 1.79)
Previous Benign Breast Biopsy	1.36	(1.02, 1.81)
Nulliparity to Age 30	0.97	(0.75, 1.24)
Age ≤ 10 at Menarche	0.82	(0.55, 1.22)
Age ≥ 55 at Menopause	0.73	(0.55, 0.97)
Age and Risk Category[b]		
Moderate Risk Category		
Age 50–59	1.00	
Age 60–79	1.86	(1.49, 2.32)
High Risk Category		
Age 50–59	3.94	(2.61, 5.96)
Age 60–79	3.09	(2.21, 4.31)

[a] Odds ratios estimated from the logistic regression model including all six risk factors and a variable for the interaction between "age" and "risk category." Odds ratios are defined so that those without the characteristic are the reference group.

[b] Odds ratios are defined so that women ages 50–59 who are at "moderate" risk are the reference group.

younger show any association with participation. Controlling for other risk factors in the multivariate model increased the association between late menopause and participation.

The association of age with participation depends on whether the risk label was "moderate" or "high." Increasing age is associated with participation only among women with the "moderate" risk label. For women labeled high-risk, participation is essentially the same or even slightly less among older women (OR = 3.09/3.94 = 0.78).

As seen in the bivariate analysis, the high-risk label shows the strongest association with participation. However, the four odds ratios for the joint factors in Table 4 reveal that the association between the high-risk label and participation was more than two times as strong in younger (OR = 3.94) compared to older women (OR = 3.09/1.86 = 1.66, Table 4).

Discussion

The results of this study substantiate our hypothesis that risk factors recognizable by lay persons such as age, family history, previous benign breast biopsy, and the high-risk label are each positively associated with compliance with a mailed recommendation to obtain a screening mammogram. Since risk factor information was collected prior to screening visits, recall bias was avoided. The prospective observation of the entire population in the course of their usual care also eliminates any Hawthorne effect created in the process of selecting and observing a separate study population.

Performing the study in the context of a formal screening program also creates some potential limitations. First, all candidates for the study were respondents to the screening survey; the relation between risk and participation might be different for non-respondents. However, since 85 percent of women complete the risk factor questionnaire, this potential threat to generalizability is expected to be minimal. Second, as a result of the screening program's design, all study women had at least one risk factor in addition to age. In considering each individual risk factor, the referent group consisted of women without any factor but possessing "other" risk factors. While it is not possible to determine from these data whether the magnitude of the associations might differ substantially if the referent population included women whose only risk factor is age, it is likely that the associations would be stronger.

A final limitation is that the results may be biased due to residual confounding by two factors. The first is socioeconomic status (SES) for which no data were available. Since higher socioeconomic status has been associated with increased risk of breast cancer[15] and with increased participation in screening,[5,7] it must be considered a potential confounder. However, any effect of SES would be expected to be small since GHC is somewhat more homogeneous with respect to SES compared to the surrounding SMSA population.[11] Second, there may be residual confounding due to the presence of breast cancer symptoms. Participants in the HIP study were more likely to report pre-menstrual breast tenderness or having previously felt a breast lump.[5] However, when women call for an appointment at the GHC screening center, the policy is to ask about pain, lumps, or the presence of nipple discharge. Women with any of these symptoms are advised to see their primary care physician, so it is expected that the majority of screened women were asymptomatic. Symptomatic women who called for an appointment and were referred to their physician would have been misclassified as non-participants in this study since the program does not document these calls in its automated data base. This misclassification would weaken the observed associations.

Our data show that family history of breast cancer is significantly associated with participation even after controlling for other risk factors. French's work found that participants were more likely to have a family history of breast cancer than non-participants, but the difference was not statistically significant.[9] In the HIP study, there was no significant difference in "family history of cancer" between women who completed all four screening examinations compared to those who did not.[5] However, that study did not evaluate the relationship between family history of breast cancer and participation.

Our finding of increased participation among older women is not consistent with previous reports which show associations in the opposite direction.[5–7] However, none of these studies included women beyond 64 years of age and only one was conducted prospectively.[7] The data presented here suggest women 60 to 79 years of age are more likely to participate than women 50 to 59 years of age. While the reason for lower participation in younger women is not clear, one possible explanation is that these women are more likely to be employed and therefore have conflicts during usual screening clinic hours.

Our finding of increased participation in older women is important because more women are living longer and the incidence of breast cancer increases greatly with advancing age. Celentano has pointed out that primary care providers often fail to offer cancer screening procedures to the elderly.[16] Others have suggested that cost is a barrier.[17,18] The relative importance of these two factors is not clear. With the advent of Medicare coverage for mammography every two years, it will be important to observe whether the use of mammography increases among older women in other populations. Our data suggest that senior women will use mammography if it is offered to them, and cost is not an issue.

No other studies to our knowledge have examined the association between participation and the three other risk factors studied here: nulliparity to age 30, early age at menarche, and late age at menopause. Our findings are not completely consistent with our hypothesis that these factors would be unassociated with participation. No associations exist in the bivariate analysis. However, after controlling for all other risk factors in the multivariate model, a negative association appears between late menopause and participation. The explanation for this is not clear, but one consideration is that there is confounding by some other factor such as estrogen use. This relation needs closer examination in future studies.

The increased participation among women with particular risk factors that is demonstrated in this study is consistent with the Health Belief Model[19,20] which suggests that perceived vulnerability to disease should influence the likelihood that a person will undertake preventive behaviors. We hypothesized that women with certain risk factors would be more likely to perceive themselves as vulnerable, and therefore participate in screening. While the increased participation has been demonstrated with our data, we did not directly evaluate women's perception of their vulnerability.

The risk assessment/feedback process used in Group Health Cooperative's Breast Cancer Screening Program is similar to Health Risk Appraisal (HRA) and may be a way to increase the use of mammography. The HRA process provides individuals with quantitative and/or qualitative disease risk estimates based on self-reported risk factors obtained through questionnaires.[21] Proponents of HRA argue that it is an effective strategy for facilitating positive health-related behavior. Our finding that women given a high-risk designation were most likely to participate suggests that this may be true. However, more needs to be learned about the effect of different risk labels, and the influence of these designations on other behaviors. Experience from hypertensive labeling suggests that there are potential negative effects such as increased depressive symptoms, and lower self-perceived health.[22]

In conclusion, overall our study found the expected positive associations between participation and those risk factors that could be easily identified by women. Self-selection for screening by women at increased risk has been identified as a major concern in the non-experimental evaluation of screening.[1–3] Our findings give substance to these concerns but also raise the possibility that informing women of all their risk factors or using a summary risk label may take advantage of the motivation behind self-selection and encourage participation.

APPENDIX
Risk Algorithm*

High-Risk Group	• Previous breast cancer • Mother with breast cancer • Age 50+ and 2 VRF listed below
Moderate-Risk Group	• Age 40–49 and 2 VRF listed below • Over age 50 and 1 VRF listed below
Borderline Risk	• Age 40–49 and 1 VRF listed below
No Increased Risk	• No VRF, any age
Variable Risk Factors (VRF)	• Previous cancer (other than breast) • First or Second degree relative (other than mother) with breast cancer • Menarche age 10 or under • Nulliparity • 1st pregnancy age ≥ 30 • Menopause age ≥ 55 • Previous benign breast disease

*Risk algorithm used in Group Health Cooperative's Breast Cancer Screening Program until April 1988.[13]

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RWJ Foundation Awards Grant to Study Child Abuse Responses in 50 States

The first 50-state survey of child abuse policies and regulations will be launched by the American Bar Association (ABA) with a grant from the nation's largest health care philanthropy. The $280,464 two-year grant has been awarded to the ABA Fund for Justice and Education from the Princeton-based Robert Wood Johnson Foundation. The ABA, in conjunction with the American Academy of Pediatrics, will develop recommendations about policies and procedures related to prevention, intervention, and investigations of child abuse.

Under the direction of Susan J. Wells, PhD, the ABA survey will gather and analyze information from child welfare agencies, physicians, coroners, police officials, prosecutors, and legislators in all 50 states to determine how state and local governments are currently responding to child abuse.

It is estimated that child deaths from abuse have increased 34 percent since 1985, resulting in about 5,000 deaths a year, said Leighton E. Cluff, MD, Foundation president. "But because states don't collect child abuse data uniformly, there is little information to guide policymakers and other officials working to prevent these deaths," he added. Cluff termed the ABA project "an important first step" in addressing the problem and in understanding the effect of interventions intended to prevent child abuse.

In addition to collecting data, the ABA will provide consultation to selected states that have expressed interest in revising their policies regarding child abuse.

The Robert Wood Johnson Foundation was established as a national philanthropy in 1972. Since then, it has awarded more than $996 million in grants to improve health care for such US populations as adolescents, children and mothers, the elderly, the homeless, the mentally ill, people with AIDS, and the uninsured. For further information, contact the RWJ Foundation, PO Box 2316, Princeton, NJ 08543-2316. Tel: (609) 452-8701.

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Breast Cancer Screening by Mammography: Utilization and Associated Factors

Jane G. Zapka, ScD, Anne M. Stoddard, ScD, Mary E. Costanza, MD, and Harry L. Greene, MD

Abstract: The status of mammography screening experience and factors related to utilization were examined in six towns serviced by physician staffs at five hospitals. Data were collected via random digit dial telephone interview of a probability sample of 1184 women, aged 45–75 years. The results showed that 55% of the women reported ever having had a mammogram. Of those who had ever had a mammogram, 21% reported that the mammogram in the past year was their first one. Of those women who are over 50 and had ever had a mammogram, 57% reported one in the past year. Analyses demonstrated that a combination of demographic factors, certain beliefs and knowledge, having a regular physician, social interaction and media exposure are independently related to ever having a mammogram, and to having one in the past year. Despite anecdotal and empirical evidence that the proportion of women ever having had a mammogram has substantially increased in the past several years, increasing utilization among older and lower-income women provides a challenge for public health (*Am J Public Health* 1989; 79:1499–1502.)

Introduction

Among women, breast cancer leads all other cancers (28% in women), and is the second leading cause of cancer death.[1] Reductions in mortality from breast cancer depend on successful interventions aimed at early detection and treatment,[2–4] but the adoption of screening technologies by physicians and women has been slow.[5–7] However, there are recent indications that the utilization of screening technologies, notably mammography, has been increasing.[8] Targeted interventions are being called for by medical and public health authorities.[4,5] This study was undertaken to characterize the breast cancer screening experience, knowledge, and attitudes of a probability sample of urban women so as to provide a valid base for planning community interventions aimed at increasing the appropriate use of screening technologies.

Methods

A survey was conducted using the Waksburg random digit dial method[9] in two urban areas of eastern Massachusetts comprising six towns serviced by physician staffs of five community hospitals. A pretested structured 20-minute telephone interview was administered in late 1987. A woman was eligible for the study if she was between 45 and 75 years of age and had never had breast cancer. A minimum of 10 call-backs were made as needed to secure an interview, and a refusal conversion process was included. A response rate of 75.3 percent was achieved, yielding a sample of 1,184 women.

In addition to sociodemographic and health history items, the survey included items to measure knowledge (such as risk factor and incidence knowledge and knowledge that mammography is appropriate in the absence of symptoms), beliefs (such as perception of barriers to and benefits of having a mammogram, and susceptibility to breast cancer), enabling factors (insurance coverage, having a regular physician and usual location of care) and factors which reinforce or hinder screening behavior (encouragement to have screening by family, friends, and providers, discussion about screening with social network members and exposure to media messages about breast cancer screening). Summary variables were computed for items purported to measure personal and social factors and tests of reliability yielded Cronbach's alpha coefficients in the range of .56 to .71.*

We first looked at variables associated with ever having a mammogram in the total sample. Among women who had a previous mammogram, we subsequently looked at correlates of having a mammogram in the last year for those aged 50 and over. While recent study results suggest reduction in mortality from breast cancer with mammography in younger women,[10] the issue remains controversial. Therefore, women under age 50 were removed from this analysis since current guidelines do not consistently recommend annual screening for women under age 50.

For this exploratory study, forward stepwise logistic regression analysis[11] was used to select from among those bivariately associated with screening, a set of variables representing independent correlates of screening behavior. After main effect variables were entered into the model, selected two-way and higher order interactions were considered. The entry criterion was a chi-square p value of 0.05. Some interactions could not be tested due to sparse cells in the relevant multiway cross classifications.

Results

Selected characteristics of the sample are presented in Table 1. Fifty-five percent of the 1184 women surveyed reported ever having a mammogram. Among the 45 percent who had never had a mammogram, 10 percent had never heard of mammography. Twenty-five percent of the total sample reported previous medical problems with their breasts and these women were significantly more likely to have had multiple mammograms than those who reported never having had a problem. However, the majority of women (82 percent) reported having had their mammogram as a "check-up, for no particular problem." Women who had less than a high school education, or were Catholic or

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*Details on the survey items, factor analyses, and development of summary variables are available from the authors.

TABLE 1—Percent (and frequency) of Reported Breast Cancer Screening Behaviors by Selected Characteristics[a]

Characteristics	Total Sample n = 1184	Ever Had a Mammogram n = 650	Mammogram in Past Year[b] n = 290
Age (years)			
45–49	222	56.8 (126)	— —
50–54	224	60.7 (136)	32.7 (73)
55–59	200	61.0 (122)	30.0 (60)
60–64	197	55.3 (109)	33.5 (66)
65–69	165	45.0 (75)	27.9 (46)
70–75	152	46.7 (71)	29.6 (45)
Education			
Less than high school	215	38.6 (83)	21.5 (41)
High school graduate	513	55.8 (286)	29.3 (125)
Some college	255	59.2 (151)	35.8 (72)
College graduate	192	65.1 (125)	43.7 (52)
Employment			
Currently employed	598	57.3 (343)	31.0 (130)
Full-time homemaker	192	56.3 (108)	30.8 (49)
Retired	286	51.1 (146)	33.3 (93)
Other	100	48.0 (48)	22.0 (18)
Income			
Less than $15,000	333	40.5 (135)	22.9 (69)
$15,001–25,000	201	57.2 (115)	28.6 (48)
$25,001–35,000	163	58.3 (95)	32.0 (40)
More than $35,000	320	66.3 (212)	42.9 (90)
Religion			
Catholic	754	51.7 (390)	27.8 (167)
Protestant	283	56.9 (161)	31.4 (74)
Jewish	83	84.3 (70)	63.3 (38)
Marital status			
Married/living with	791	57.7 (457)	32.9 (203)
Separated/divorced	130	51.5 (67)	25.0 (22)
Widowed	211	46.9 (99)	25.8 (51)
Never married	43	48.8 (21)	42.4 (14)
Ever had medical problem with breast			
Yes	292	83.6 (244)	43.6 (98)
No	892	45.5 (406)	27.0 (192)

[a] Frequencies may not sum to total sample due to missing data in respective cross-tabulations.

[b] For women over 50 years of age.

Protestant (as compared to Jewish), were significantly less likely to have ever had a mammogram or had one in the past year. Income was directly related to ever having a mammogram; education and employment were highly correlated with income for the 86 percent of subjects who responded to the income question.

Given that getting a mammogram requires interaction with the medical care system, we were particularly interested in several variables related to sources and perceptions of medical care as well as insurance variables. Eighty-five percent of women reported that they had a regular physician. A majority (74 percent) of those who had a mammogram had had a clinical breast examination in the last year vs 44 percent of those who had never had a mammogram. Seven percent of the sample did not have any level of health insurance or entitlements. Seventy-one percent of those who ever had a mammogram said their insurance paid for it, 10 percent paid for it themselves, and 16 percent reported some combination.

Women who had never had a mammogram were asked why. Forty-five percent responded that they did not feel it was necessary and/or that they felt healthy, and 24 percent said their doctor had never recommended it. All women were asked if they would have a mammogram in the next year if their doctor recommended it, and 83 percent said they were very certain they would.

Factors Associated with Mammogram Experience

Table 2 presents the results of the main effects logistic regression analysis on ever having had a mammogram. There were significant negative interactions (data not shown) between being encouraged by a friend to have a mammogram and disagreeing with the statement that symptoms should precede a mammogram, and between being encouraged by a friend and discussing mammography with a friend. These interactions indicate that once one event has occurred there is no additional association with the other event. Variables which showed bivariate associations with ever having had a mammogram but which did not enter the model include knowledge of breast cancer risk factors, perceptions of susceptibility to breast cancer, perception of barriers to mammography, a belief that other health problems have priority, and education attainment. Women whose regular physician was an internist or gynecologist were more likely to report ever having a mammogram than women whose physician was a general or family practitioner (68 percent vs 41 percent); the latter women were no more likely to have had a mammogram than women with no regular doctor.

The women age 50 and over who had ever had a mammogram were divided into those who had one within the past year and those who had not. The main effects logistic regression model for this comparison is given in Table 3. Although having a mother or sister with breast cancer was positively associated with ever having a mammogram, having another relative (e.g., aunt) with breast cancer was negatively associated with having one in the last year. Variables which showed bivariate associations with mammography in the last year but which did not enter the model include a belief that mammography can detect cancer, history of breast problems, and employment status.

We were interested in the impact of insurance coverage on mammogram experience. Reliable and valid information on coverage was limited. A substantial number of subjects reported having more than one type of coverage. Since it was not possible to determine which plan provided coverage,

TABLE 2—Factors Associated with Ever Having a Mammogram: Main Effects Logistic Model (n = 840)

Variables	B	Odds Ratio	(95% CI)
Sociodemographic			
Income	—		
$15001–25000	.54	1.72	(1.08, 2.75)
$25001–35000	.03	1.03	(0.62, 1.70)
$35001+	.43	1.53	(1.00, 2.35)
Religion			
Catholic	−.09	0.91	(0.63, 1.33)
Jewish	1.14	3.14	(1.30, 7.60)
Health History			
History of breast problems (yes)	1.69	5.40	(3.47, 8.41)
Mother/sister with breast cancer (yes)	.95	2.59	(1.43, 4.68)
Knowledge			
Need symptom (disagree)	.76	2.15	(1.75, 2.63)
Attitude/Belief			
Benefits of mammography (agree)	.16	1.17	(1.08, 1.28)
Enabling			
Have a regular MD (yes)	.75	2.13	(1.30, 3.48)
Reinforcing			
Mammogram encouraged by a friend (yes)	.62	1.85	(1.20, 2.87)
Discuss mammography with friends (yes)	.71	2.04	(1.43, 2.89)
Constant	−2.38		

Hosmer-Lemeshow Goodness of fit: Chi-square = 5.78, df = 8, p = .67.

TABLE 3—Factors Associated with Having Had a Mammogram within the Past Year for Women Over Age 50: Main Effects Logistic Model (n = 346)

Variables	B	Odds Ratio	(95% CI)
Sociodemographic			
Income			
$15001–25000	−.67	0.51	(0.25, 1.06)
$25001–35000	−.34	0.71	(0.32, 1.61)
$35001+	.56	1.75	(0.78, 3.95)
Marital			
Separated/divorced	.41	1.50	(0.60, 3.79)
Widowed	−.70	0.50	(0.23, 1.08)
Never married	2.14	8.46	(1.25, 57.28)
Age (years)			
60–69	.99	2.69	(1.48, 4.86)
70–75	1.65	5.22	(1.88, 14.45)
Health History			
Mother/sister with breast cancer (yes)	.88	2.40	(1.06, 5.44)
Other blood relatives had breast cancer (yes)	−1.01	0.36	(0.17, 0.78)
Reason for mammogram (routine)	.84	2.31	(1.22, 4.38)
Knowledge			
Need symptom (disagree)	.37	1.45	(1.03, 2.04)
Know cost (yes)	.84	2.31	(1.09, 4.91)
Attitude/Belief			
Most women over 50 get a mammogram (agree)	.37	1.45	(1.09, 1.91)
Enabling			
Have regular MD (yes)	1.10	3.02	(1.09, 8.39)
Mammogram satisfaction (high)	.16	1.17	(1.01, 1.36)
Reinforcing			
Media exposure (greater)	.92	2.51	(1.11, 5.72)
Constant	−2.32		

Hosmer-Lemeshow Goodness of fit: Chi-square = 6.14 df = 8, p = .63.

women with more than one type of coverage were excluded and the screening behavior of those women who reported only one type of coverage was further examined. Given the possibility that a person's insurance plan changes over the years, analysis was limited only to mammogram experience in the past year. Bivariate analysis demonstrated that 34 percent of women who had commercial insurance, health maintenance organization (HMO) or Medicare plus commercial insurance had a mammogram in the past year compared to 15 percent of women covered by Medicare only, Medicaid only, or no insurance. For this set of subjects, the insurance variable did not significantly improve the multiple logistic model.

Discussion

The findings of this study support anecdotal and empirical evidence that the proportion of women ever having a mammogram has substantially increased in the last several years, particularly among White, middle and upper socioeconomic level, and younger women.[5,8,12] Further, we found that although a greater proportion of women over age 60 have never had a mammogram, among those who have ever had one, more older women than younger women did get one in the past year. A summary of data from 33 states[13] reported that 29 percent of US women 50 years of age or older reported having had a screening mammogram in the last year. The level of screening varies considerably by state, however, with 37 percent reported in Massachusetts.[14] For our sample of women ages 50–75 years, 31 percent reported a mammogram in the past year.

It is very difficult to determine the extent to which women have had mammographic examinations for screening rather than diagnostic purposes.[5] Our data demonstrate a positive correlation between previous breast problems and having had one mammogram or having multiple mammograms. However, 82 percent of the women reported that their most recent mammogram was for "check-up" purposes, not for a particular problem. Additionally, there is uncertainty in estimating the proportion of women who have been screened with mammography at some time in their lives as compared with the proportion who are routinely screened according to recommended age/frequency guidelines. Defining the reasons for mammography and documenting the prevalence of *regular* screening should be priority questions for prospective research on screening utilization.

The regression models reported here must be interpreted with caution. Stepwise forward selection of variables was used as a means of screening many bivariately associated variables to select those independently associated with screening behavior. Although this approach is suitable for screening it does not necessarily result in valid estimates of effect size.[15] The results of this analysis may be used to develop specific models which can then be explicitly estimated and tested in another data set.

Perception of barriers to mammography (exposure to radiation, pain, cost, and embarrassment) and to breast cancer screening have been suggested by theoretical models[16] and emphasized in previous studies.[17,18] Interestingly, none were significantly related to mammography behavior. Neither perception of severity, nor risk factor knowledge were associated. In contrast to the limited relationship of knowledge and belief factors, importance of having a regular physician, particularly a gynecologist and/or internist, is highlighted in this research.

Although the findings of this study cannot be generalized to minority or rural populations, they do highlight a particular challenge for public health to increase screening utilization behavior in older and lower-income women. Targets for education include their clinicians who need to initiate more effective discussion of screening requirements, motivation of public policy-makers to include coverage of screening mammograms to deal with any cost/access barrier, as well as education of women about the need to attend to breast health.

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Editor Search Committee Seeks New Editor for American Journal of Public Health

An Editor Search Committee is actively recruiting a replacement for Dr. Alfred Yankauer who has announced his retirement as Editor of this *Journal*, effective July 1, 1990. The six-member search committee welcomes and solicits nominations and recommendations from the APHA membership and leadership to assist them in identifying the best available individual for this important salaried position. Nominations should be submitted as soon as possible, as the committee will meet several times to consider suggestions, applications, and support materials of potential candidates.

Names of potential candidates, along with letters of endorsement and other support materials, should be sent to: Editor Search Committee, American Public Health Association, 1015 15th St., NW, Washington, DC 20005.

The following criteria will be considered by the search committee in selecting the Journal editor:

- Comprehensive knowledge and broad perspective of the field of public health, with an appreciative understanding of its many disciplines and solid grounding in the basic sciences of epidemiology and statistics;
- Professional accomplishment and identity with the public health field, including respected standing among peers, combined with an extensive network of professional contacts who will provide expertise in soliciting and evaluating materials for publication;
- Demonstrated research skills, with evidence (such as credited publication in peer review journals) of firm grounding in a field of scientific inquiry within public health;
- Demonstrated writing, reviewing, and editing skills, enabling authoritative advice to authors on the suitability of prepared manuscripts, facilitating informed consideration of reviewer assessments, and equipping the preparation of appropriate editorials as needed;
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Nulliparity, Decade of First Birth, and Breast Cancer in Connecticut Cohorts, 1855 to 1945: An Ecological Study

ROBERT A. HAHN, PHD, MPH, AND SURESH H. MOOLGAVKAR, MD, PHD

Abstract: Risk of breast cancer increases with age at first birth, and is lower in women who bear their first children while young than in nulliparous women. While previous studies have investigated risk of breast cancer in birth cohorts by examining partial aspects of cohort childbearing, the present ecological study assesses total cohort childbearing risk in Connecticut women born between 1855 and 1945. In each cohort, the proportion of women nulliparous and first bearing children in their twenties, thirties, and forties are weighted by relative risks associated with these events as ascertained in previous studies. Summed cohort childbearing risks are compared to the incidence of breast cancer in women 40 years of age and older in the same cohorts. Changes in decade of first birth and nulliparity do not explain the changes in breast cancer incidence observed: while cohort childbearing risk has declined over the period examined, breast cancer incidence has increased in the same cohorts. Alternative explanations for cohort increases in breast cancer incidence are reviewed. (*Am J Public Health* 1989; 79:1503–1507.)

Introduction

In the United States, Europe, and elsewhere, breast cancer incidence and mortality reached a low point in women born at the turn of the twentieth century and have been rising steadily in cohorts born thereafter.[1,2] The predominance of cohort effects in accounting for these secular changes in breast cancer incidence and mortality was noted early by MacMahon,[3] and has been well established by the modeling methods of indirect standardization.[1,2,4,5] MacMahon hypothesized that these cohortwise-changes in incidence and mortality might correspond to changing patterns of childbearing.[3]

With several exceptions,[6–8] most case-control studies[9–13] have demonstrated that a woman's risk of breast cancer increases with the age at which she completes her first full term pregnancy. The risk of breast cancer in women first bearing a child before age 18 is estimated to be one-third that of women first bearing after age 34. Nulliparity is associated with greater risk of breast cancer than bearing a first child up to age 34, after which the risk associated with first births exceeds that of nulliparity.[13] A large retrospective cohort study[14] further supports these results.

Several studies[7,15–20] have examined the association of changing population childbearing patterns with changing patterns of breast cancer incidence and mortality. Using summary vital records from England and Wales, Connecticut, and the United States, MacMahon[15] examined the association of cohort fertility and breast cancer incidence and mortality; findings contradicted the hypothesis that fertility was inversely associated with breast cancer rates. In contrast, Armstrong[16] examined proportions of women in the US bearing first children by certain ages and live birth rates by certain ages in England and Wales; he found that childbearing trends accounted at least in part for the cohort variation in breast cancer incidence (in Connecticut) and mortality (in the US and in England and Wales). Likewise, Blot[17] found parallels between proportions of US women nulliparous between ages 20–24 and breast cancer mortality in the same cohorts. Wigle[18] also demonstrated associations between marital and fertility characteristics of Canadian women and their rates of breast cancer incidence and mortality. On the other hand, based on correlations among rates of breast cancer mortality among single and married women in nine nations and regions within them, Hems and Stuart[19] claimed that international differences in breast cancer mortality were associated with diet but not with fertility. Most recently, White[20] predicted that delayed childbearing in cohorts of women born since 1945 will yield breast cancer incidence significantly greater than that of preceding cohorts, in which early childbearing was more common.

Other than White's predictive analysis, these studies have shortcomings of two sorts. Some[7,15] have examined risk factors tangential to those now recognized to be associated with rates of breast cancer, for example, cohort fertility by older ages, and proportions married at 20–24 and at 40–44 years. Further, all of these studies examine one or several aspects of cohort childbearing events, e.g., nulliparity between ages 20 and 24, first birth by certain ages, but not the overall cohort history of childbearing. Investigation of isolated elements of cohort childbearing may mask other cohort childbearing risk factors for breast cancer which either support or oppose the particular cohort risk factor investigated. For example, while one cohort may have apparently greater risk than another cohort because of high nulliparity in ages 20–24, it may also have lower risk than the other cohort because of a high rate of first births to women younger than 20 years. Apparent associations and non-associations between partial cohort childbearing events and breast cancer outcomes may thus be misleading.

The present study used vital records and Connecticut Tumor Registry data to examine the association between total cohort childbearing history—nulliparity and decade of first birth—and breast cancer incidence in the population of Connecticut women born between 1855 and 1945.

Methods

Information on cohort childbearing events was abstracted from the Connecticut Vital Statistics Report (1879–1945),[21] Vital Statistics of the United States (1946–1982),[22] and the US Census of Population (1880–1980).[23] Information on breast cancer incidence in Connecticut women, ages 20 to 79 years, from 1935 to 1982 was provided by the Connecticut Tumor Registry,[24] as reported in Stevens, *et al*,[2] and updated.

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Cohort Decades of Firth Birth and Nulliparity

Because Connecticut Vital Statistics Reports present information on women bearing their first children in time intervals of varying length, but commonly in 10-year age categories, e.g., between ages 20 and 29, the present investigation is constrained to a comparison of decades of first birth. Numbers of women bearing first children in each year were aggregated into 10-year groupings centered on decade years (in which Census data were gathered), e.g., 1885–1894 (with decade mid-year 1890), 1895–1940 (mid-year 1900), etc. Since Connecticut Vital Statistics Reports were first published only in 1879, information on first births at different ages in the 1880 "mid-year" was aggregated from 1879 to 1884.

Proportions of women bearing first children in their second, third, fourth, and fifth decades were calculated by dividing numbers of first births in each age group by the population of that group. Since vital registry data on first births for the first Census period (1880) were available only for six of the 10 years in one interval (1879–1984), the proportions of 2nd to 5th decade first births for this period were estimated by dividing the numbers of births by 6/10 of the appropriate Census figures for each age group.

Resultant proportions were reorganized according to the year of birth of the mothers. Thus, proportions of women who bore a first child in their second decade (i.e., ages 10–19) between 1879 and 1884 were associated with those who first bore in their third decades (i.e., 20–29) between 1885 and 1894, and with those who first bore in their fourth decades (i.e., 30–39) between 1895 and 1904, and so on; all of these women were born between 1855 and 1874, a 20-year span referred to by its mid-year as the cohort of 1865.

While a few women were reported to bear first children after age 50, for the purposes of this analysis it was assumed that a women who had not borne a first child by age 50 was nulliparous. Cohort nulliparity was calculated by subtracting from *one* the sum of proportions of cohort women who had borne a first child in their second through fifth decades. That is,

$$\text{Cohort Nulliparity} = 1 - (FB_2 + FB_3 + FB_4 + FB_5),$$

where FB_n = proportion of cohort women bearing a first child in their n^{th} decade.

Cohort Childbearing Breast Cancer Risk

Cohort proportions of women nulliparous or first bearing children in different decades were weighted by the relative risks ascribed to these childbearing events by the multination case-control study of MacMahon, *et al*:[10]

Age/First Birth:	Nulliparous	<20	20–24	25–29	30–34	>34
Risk	1.0	.48	.59	.76	.91	1.10

In order to make the relative risks of five-year age groups from the multi-nation[10] study commensurate with the decades of first birth in the present study, a mean of relative risks within decades was taken to estimate the relative risk for each decade. Since women older than 40 contributed less than 0.5 percent of cohort first births, their contribution to childbearing risk was no longer considered. The total nulliparity and first birth decade risk of breast cancer in cohorts was then estimated by summing the relative risks specific to each childbearing category, weighted by the proportions of each childbearing category in the cohort. That is,

$$\text{Childbearing Breast Cancer Risk} = FB_2 \times RR_2 + FB_3 + FB_4 \times RR_4 + \text{Proportion Nulliparous} \times RR_{\text{Nulliparity}},$$

where, as above,

FB_n = the proportion of cohort women bearing a first child in their n^{th} decade,

and where,

RR_n = the risk of breast cancer given this event, relative to nulliparity.

Breast Cancer Incidence and Standardized Incidence Ratios

Since the first birth "exposures" considered were those occurring to cohort females by age 40, only breast cancer incidence after this age was analyzed as a possible outcome. Incidence data for women older than 40 years were grouped into 10-year age categories, and arrayed by cohort to parallel available information on childbearing exposures. The number of cases divided by the corresponding sum of woman-years at risk gave the incidence of breast cancer in this group during this period.

To compare cohort experience of breast cancer incidence and changes of breast cancer incidence between cohorts, we computed an aggregate incidence measure for the cohort. Since age-specific data are missing in the youngest cohorts, the usual methods of direct and indirect standardization do not apply. Following the method of Breslow and Day,[1] we computed an internally standardized incidence ratio for each cohort. The method assumes that age effects are constant in all cohorts, and cohort effects constant for each age; both parameters are estimated for all ages and all cohorts by assuming their constancy in cells for which no data are available. It is assumed that the populations are large, and that disease events are rare and well characterized by the Poisson distribution. Further, the expected number of events (incident cases of breast cancer) in age group i and birth cohort j is assumed to be $E_{ij} = N_{ij} \times a_i \times b_j$, where N_{ij} is the population (woman years) at risk in age group i and cohort j, a_i is the effect of age group i, and b_j is the effect of birth cohort j. The parameter, b_j, can be thought of as an internally standardized incidence ratio. Maximum likelihood procedures are then used to estimate the parameters of the model. This method yields a summary incidence ratio for cohort data derived from cross-sectional studies.

Results

Cohort Childbearing Breast Cancer Risk

For all cohorts of Connecticut females born between mid-years 1865 and 1935, the largest proportion of women have borne a first child in their third decade; the second largest proportion are nulliparous, except for women born in mid-year 1935; the smallest proportions have borne first children in their 2nd or 4th decade (Table 1). Together, cohort proportions of women either nulliparous or first bearing children in their third decades account for between 72 percent and 83 percent of cohort women. Predominant cohort trends are the large, though irregular, increase in the proportion of women bearing their first children in their twenties and the correspondingly large decline in nulliparity.

Weighting of cohort childbearing proportions by the corresponding relative risks yielded a cohort childbearing breast cancer risk profile (Figure 1) indicating: a general declining trend from earlier to later cohorts, interrupted by a small increase from the cohort of 1865 to that of 1875, and a larger increase from the cohort of mid-year 1895 to that of 1905. These risk estimates correspond mainly to changing proportions of third decade first births and nulliparity. Similar

TABLE 1—Proportions of Connecticut Women Nulliparous and First Childbearing in Different Decades (2nd–4th) by Birth Cohort Mid-Year, 1865–1935

Cohort Mid-Year of Birth	Nulliparas	Woman's Age at First Childbirth 10–19	20–29	30–39
1865	0.33	0.11	0.46	0.09
1875	0.36	0.10	0.44	0.09
1885	0.28	0.10	0.51	0.10
1895	0.26	0.12	0.53	0.08
1905	0.30	0.13	0.43	0.14
1915	0.21	0.09	0.55	0.15
1925	0.13	0.09	0.70	0.08
1935	0.10	0.14	0.70	0.06

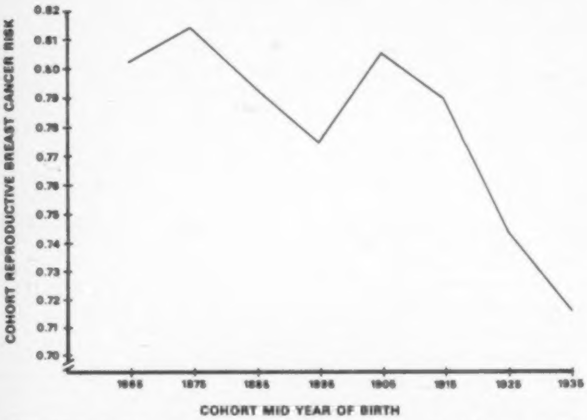

FIGURE 1—Childbearing Breast Cancer Risk in Connecticut Women, 1855–1945

breast cancer childbearing risk profiles (not shown here) derive from application of the relative risks from the large cohort study of Tulinius, *et al*,[14] and from the meta-analysis of White.[20]

Breast Cancer Incidence and Age-Adjusted Incidence Ratios

Incidence among women age 40 and older has increased steadily, with possible interruption in the cohort of 1885 for 70 year old women, of 1895 for 60 year old women, and of 1935 for 40 year old women (Figure 2). (The study of Stevens, *et al*,[2] indicated a trough in the standardized incidence ratio between Connecticut cohorts of 1885 and 1895 and a possible decline following the cohort of 1925.)

The standardized incidence ratio in women 40 years and older showed a general increase from the earliest to the last cohorts (Figure 3). Only from the cohort of 1905 to that of 1915 was there a slight decline in the incidence ratio, followed again by an increase—a more gradual increase than that preceding the decline. Points of inflection showed a rather different pattern from Figure 11 in Stevens, *et al*,[2] principally because Stevens' model incorporated year of event (that is, the interaction between age and cohort). In addition, whereas Stevens, *et al*, considered women 20 years and older in five-year age and cross-section categories, we considered only women 40 years and older in 10-year age categories and 10-year cross-sections. Our study also includes three years of more recent data.

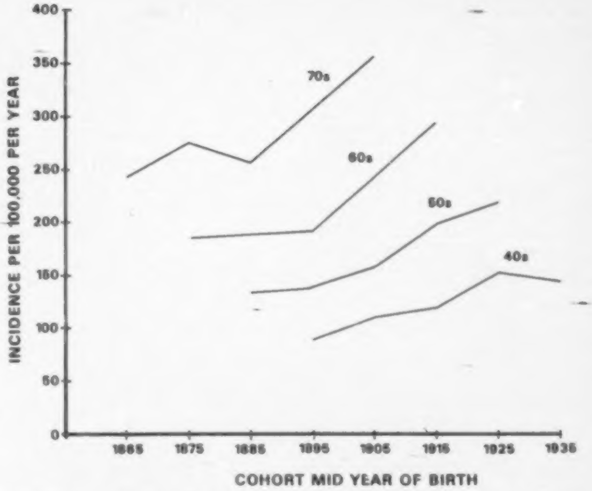

FIGURE 2—Breast Cancer Incidence in Connecticut Women Ages 40–79, by Mid-Cohort Year of Birth

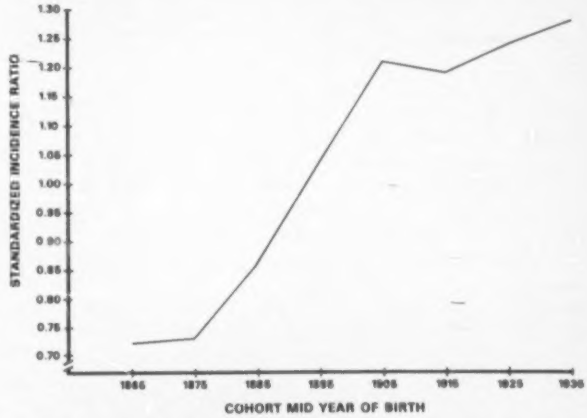

FIGURE 3—Standardized Incidence Ratio of Breast Cancer for Connecticut Women, 40 Years, by Mid-Year of Birth Cohort

Childbearing Breast Cancer Risk and the Standardized Incidence Ratio

Comparison of cohort childbearing breast cancer risk (Figure 1) and the standardized breast cancer incidence ratio for the same cohorts (Figure 3) indicated clearly opposing trends. While childbearing proportions of cohorts would have predicted a general fall in incidence, a generally increasing trend was observed in the standardized incidence ratio. Indeed, higher predicted childbearing breast cancer risk was associated with lower actual incidence ratios, and vice versa.

Comparison of childbearing breast cancer risk (Figure 1) with age-specific incidence rates (Figure 2) also showed a general opposition of trends.

To review the comparisons made in prior population based studies[7,15–20] with evidence from the present data, we compared the standardized incidence ratio with each of the childbearing proportions—nulliparity, 2nd, 3rd, and 4th decade first births. Discernible relations were contrary to those hypothesized. For example, the proportion of 4th decade first births was unrelated to the standardized incidence ratio, and the proportion of nulliparas was inversely related.

Discussion

This ecological study examines the association of basic reproductive events—childbearing and decades of first birth—with subsequent risk of breast cancer for all Connecticut women born between 1855 and 1945. Predicted changes are contrary to those observed. While changing childbearing patterns predict a general decline of breast cancer incidence from the cohort of 1865 to that of 1935, the standardized breast cancer incidence ratio generally increases.

These findings are consistent with the large population-based cohort study of Tulinius, *et al*,[14] in which nulliparity and age at first birth were significantly associated with breast cancer incidence *within* cohorts, but predicted incidence changes *between* cohorts contrary to those observed. It may be noted that international differences in childbearing patterns, e.g., nulliparity and early first births, likewise cannot explain the large international differences in breast cancer incidence or mortality, for example, the 4- to 5-fold differences between the US and Japan.[25,26] In fact, breast cancer mortality among US males is also roughly four times that of Japanese males, suggesting that factors unrelated to gender and childbearing history are important in determining international differences.[27]

The conclusions of the present study do not contradict the association of nulliparity and age at first birth with breast cancer incidence. They suggest, however, that changes in breast cancer incidence among cohorts cannot be explained on the basis of changes in cohort childbearing. Cohortwise increases in breast cancer incidence, not only in Connecticut but elsewhere also,[2] have not been accounted for.

Several other recognized risk factors merit further consideration.

Age at Menarche and at Menopause: Early menarche and late menopause are recognized risk factors for breast cancer.[13] While age at menopause has remained relatively constant over the last century,[28] age at menarche has declined at a rate estimated at two months per decade,[29] thus suggesting an increase in breast cancer incidence.

Oophorectomy: Oophorectomy has been shown to be associated with subsequent decreased risk of breast cancer.[13] Armstrong[16] and others have attempted to show a negative association between rates of hysterectomy and breast cancer incidence. This comparison is misleading since it is not hysterectomy, but associated oophorectomy which affects breast cancer risk reduction. Changing rates of oophorectomy have not been examined for periods prior to 1960,[30] but this operation is probably rare and relatively recent, and thus is unlikely to be associated with the breast cancer changes considered here.

Oral Contraceptive Use: The effect of oral contraceptive use on subsequent breast cancer is a matter of controversy. Pike, *et al*,[31] have found that oral contraceptive use prior to first full term pregnancy increases the risk of breast cancer in young women (younger than 32 years). Vessey, *et al*,[32] and others[33] find no such effect. Despite their current prevalence, the recency of oral contraceptives precludes their association with the secular changes considered here.

First Trimester Abortion: The association of abortions prior to first full term pregnancy with increased risk of breast cancer in young women is also a matter of controversy[32,33] Documentation of abortion prior to the 1973 Supreme Court decision is problematic, but this procedure has probably become widespread only recently, and thus again is unlikely to be associated with the changes considered here.

Multiparity: While multiparity and age at birth beyond the first have been found to have less effect on breast cancer than age at first birth,[10,13] studies of high parity populations indicate a strong effect.[14,34,35] In the US, rates of parity of two or more have generally increased since 1900,[36] suggesting a decline in breast cancer rates.

Lactation: While the international studies of MacMahon, *et al*,[37] had shown that, after controlling for parity, the effects of lactation in reducing breast cancer risk disappeared, recent studies again indicate an association, but principally in premenopausal women.[38,39] It is not clear how long-term secular changes in lactation could be assessed.[40]

Diet and Body Weight: Diet and one of its correlates, body weight, have been associated with international differences and secular changes in breast cancer rates.[7,41] Historical records of national and per capita consumption of different food products may allow exploration of the association of nutritional changes and changes in breast cancer incidence.

Electricity and Uninterrupted Light: There is experimental evidence in the rat that exposure to electro-magnetic fields or light reduces pineal production of melatonin; in turn; decreased production of melatonin leads to increased ovarian estrogen and pituitary prolactin production. Stevens[42] has hypothesized that increased exposure to light and electromagnetic fields may increase the risk of breast cancer in humans. Thus, it is interesting to speculate that increasing electrification may be responsible for some part of the observed secular trend. However, it is highly unlikely that the effect of electrification would be seen as changes in risk by cohort.

Secular trends and international differences in this common disease remain largely unexplained. The present study provides further evidence that prominent childbearing events in cohorts predict changes in cohort breast cancer contrary to those which have occurred. Other risk factors must counteract these cohort childbearing trends. Efforts should be made to identify modifiable risk factors which might explain cohort increases in breast cancer incidence.

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NH Law Center Announces Plan to Publish Interdisciplinary Journal

Franklin Pierce Law Center is launching a new interdisciplinary quarterly journal, *Risk: Issues in Health & Safety*. Manuscripts dealing with *basic* problems in health and safety management are being sought for future issues. Of particular interest are research reports or essays addressing strategies for reducing net risk or focusing on the normative issues involved in determining the acceptability of risk—particularly ones spanning traditional categories of occupational, environmental, medical, and consumer injury.

Comments, book reviews, announcements concerning events of interest to a broad interdisciplinary audience, and letters are also welcome.

Those who wish to receive a complimentary copy of the first issue, to be published in late fall or early winter of 1989, may write to Carol Ruh, Franklin Pierce Law Center, 2 White Street, Concord, NH 03301. Annual institutional subscriptions are $50; individual subscriptions, $25.

Thomas G. Field, Jr., JD, Professor of Law and founding member of the faculty of Franklin Pierce Law Center, will serve as editor of the new journal. Those interested in submitting a manuscript may send them to him at the Law Center address, or telephone (603) 228-1541 for further information. FAX Telecopier: (603) 224-3342. The Law Center also publishes *Idea: The Journal of Law and Technology*.

3T

Late-Stage Diagnosis of Breast Cancer in Women of Lower Socioeconomic Status: Public Health Implications

THOMAS A. FARLEY, MD, AND JOHN T. FLANNERY, BS

Abstract: To assess the success of breast cancer control activities in Connecticut, we examined data from the Connecticut Tumor Registry, determining differences in breast cancer stage at time of diagnosis over time and in selected subgroups. From 1982 to 1985, the percentage of women with cancer confined to the breast increased from 54.0 percent to 61.3 percent. During 1984 and 1985, lower socioeconomic status (SES) women with breast cancer were less likely than higher SES women to be diagnosed with early-stage disease (56.9 percent vs 62.7 percent). SES was estimated by census tract of residence. In the same years, the overall incidence of breast cancer was greater in higher SES women. Projections based on these incidence data found that lower SES women, as compared to higher SES women, had a higher rate of expected breast cancer deaths (24.6 vs 19.7 per 100,000), and a greater percentage of those deaths considered preventable by early detection (22 percent vs 11 percent). The rate of preventable deaths in lower SES women was 2.5 times as great as that for higher SES women (5.3 vs 2.1 per 100,000). Tumor registries can serve as useful surveillance systems to aid cancer control programs. Breast cancer early-detection programs should give special attention to lower SES women. (*Am J Public Health* 1989; 79:1508–1512.)

Introduction

The long-term survival of women with breast cancer is dependent on the stage of disease at the time of diagnosis.[1] Attempts to control breast cancer deaths have therefore relied on promoting early cancer detection and treatment. Prospective studies have demonstrated the success of screening programs in both detecting cancers in early stages and decreasing breast cancer mortality.[2–5] However, these studies have been in groups of women enrolled in health maintenance organizations or recruited for special projects. In Connecticut, women have primarily been encouraged to have breast cancer screening by their individual physicians and through educational programs of the American Cancer Society. The extent to which these efforts have been successful in the general population of the state is unclear.

The success of the breast cancer control programs should be measurable initially as an improvement in the percentage of all breast cancers diagnosed at an early stage and ultimately as a decrease in the incidence of late-stage disease. Tumor registries regularly contain information on cancer stage, and can consequently serve as surveillance systems to guide cancer control activities. We examined recent trends in breast cancer stage at the time of diagnosis in the Connecticut Tumor Registry. We then evaluated cancer stage in selected subgroups of the population, and projected estimates of the number of deaths preventable by earlier detection in women of various socioeconomic groups.

Methods

The Connecticut Tumor Registry has collected information on all Connecticut cases of invasive breast cancer and carcinoma-in-situ in a consistent manner since 1941.[6] The cancer stage is determined for each case by trained Medical Record Technicians at the Connecticut Department of Health Services (DHS) from detailed clinical, surgical, and histological reports. The staging system for breast cancer, unchanged since the registry began, can be summarized as follows: 1) *carcinoma-in-situ*; 2) *local*—invasive cancer localized to the breast; 3) *regional*—cancer in the breast with spread to regional lymph nodes or pectoral muscles; 4) *remote*—presence of distant metastases. The tumor registry has contained basic demographic information since its inception, but data on census tract of residence, which allow an estimation of socioeconomic status, have been included only since 1984.

The outcome measured was the distribution of cancer stage at the time of diagnosis. Comparisons of stage distribution were made by year from 1975 to 1985; for 1984 and 1985 comparisons were also made by race, place of residence, and socioeconomic status (SES), as estimated from census tract information. Census tract coding was not possible for 778 (17 percent) of 4,524 women with cancer diagnosed during those years. Data from these women were analyzed separately to test for potential bias the incomplete coding may have caused. We analyzed three different markers to identify census tract socioeconomic level (as determined in the 1980 census): 1) median household income; 2) percentage of persons below the poverty line; and 3) percentage of adults who have completed a high school education. These three variables were closely correlated, and the various definitions of census tract socioeconomic level led to identical conclusions; the data presented use the percentage of census tract adults who were high school graduates as the sole SES marker. Women were grouped into quartiles. The lowest SES quartile contained census tracts with 15–61 percent of adults who were high school graduates. The middle two quartiles were combined to form the middle SES group, which contained census tracts with 62–82 percent of adult high school graduates. The highest quartile contained census tracts with 83–97 percent of adult high school graduates. For analysis of Black women stratified by SES, the middle and higher SES groups were combined because of the small number of cancers in each stratum.

Population denominators for calculations of incidence trends from 1975 to 1985 were linear estimations based on 1970 and 1980 census figures for each age group. Incidence rates by SES were calculated using census tract population data from the 1980 census. Age-adjustment was done by the direct method, using the 1980 Connecticut population as the standard.

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The projected number of breast cancer-caused deaths in a cohort of women with breast cancer was derived according to the following general principle:

> Breast cancer-caused deaths = (Expected survivors in breast cancer cohort from actuarial estimates) − (Expected survivors in breast cancer cohort given presence of breast cancer)

The terms of this equation were calculated using: 1) 5-year survival rates by age and race from 1983 life tables; 2) 5-year relative survival rates for each stage established nationally[1] and in Connecticut; 3) estimates of the effect of SES on stage-specific 5-year relative survival rates from published studies;[7–9] and 4) mean annual numbers of breast cancer cases by age, race, and SES group during 1984–85 (see Appendix). "Nonpreventable" deaths represent similar estimates, assuming that the stage distribution in each age group achieved a goal as follows: *carcinoma-in-situ*—10 percent, *local*—60 percent, *regional*—25 percent, *remote*—5 percent of the total. This goal was felt to be reasonable based on published studies of breast cancer screening[5,10] and the current trend in Connecticut. "Preventable" deaths were defined as the difference between projected deaths and "nonpreventable" deaths. To help interpret these numbers of deaths, we calculated projected death rates by dividing them by the number of women in the population from which the cancer cases arose; population denominators were taken from the 1980 census. To study the longer-term impact of early detection, we projected 10-year death rates with similar methods.

We then assessed the reliability of these deaths rate estimates by varying the assumptions used, including: changing the program "goals", using different estimates of relative survival rates, and eliminating the differences in stage-adjusted survival by SES group. Changing these assumptions altered the magnitude of the projected death estimates, but caused only very small relative differences, and produced no difference in the conclusions regarding the SES group at highest risk. In general, increases in 5-year survival rates led to increases in the expected beneficial effect of early tumor detection, and increases in the ratio of projected preventable deaths in higher versus lower SES women. The data presented use 5-year survival rates established nationally (which include data from Connecticut)[1] and 10-year survival rates from the 1975–77 Connecticut breast cancer cohort.

The statistical significance of trends in disease stage was evaluated using extensions of the Mantel-Haenszel chi-square test for trends with multiple levels of exposure and multiple levels of disease.[11]

Results

Between 1975 and 1985, 21,131 breast cancers were diagnosed in Connecticut women. For 20,165 (95.4 percent) cancers, the available information was sufficient to accurately determine stage. In individual years, cancers of unknown stage represented between 3.9 percent and 6.0 percent of all cancers, with no increasing or decreasing trend. The trend of cancer stage among those cases in which information was complete is presented in Table 1. Between 1975 and 1981, there was little variation from year to year in stage at diagnosis, with no overall trend ($p = 0.29$). During this time, the percentage of cancers of known stage that were confined to the breast (*carcinoma-in-situ* and *local* stages) ranged from 52.2 percent to 55.9 percent. However, from 1982 to 1985 this percentage increased from 54.0 percent to 61.3 percent ($p = 0.0001$). Over these four years, the age-adjusted incidence of cancer in later stages (*regional* and *remote*) decreased slightly, from 5.06 to 5.02 cases per 10,000, while the breast cancer incidence overall increased from 11.6 to 13.6 and the incidence of disease confined to the breast increased from 5.9 to 8.0 cases per 10,000 (Figure 1).

During 1984 and 1985, the years in which information on SES was available, cancer stage was more closely and consistently related to SES than area of residence. There were no differences in the distribution of cancer stage among women residing in different counties. However, in cities of greater than 100,000 population (which have a disproportionately large share of lower SES women) only 56.7 percent of women presented with tumors confined to the breast, compared to 61.0 percent of women in smaller cities and towns ($p < 0.001$). Table 2 shows that women in the lower SES group, compared to women in the higher SES group, were less likely to have *carcinoma-in-situ* (6.2 percent vs 8.5 percent) or cancer confined to the breast (56.9 percent vs. 62.7 percent), and more likely to present with *remote* disease (8.9 percent vs 5.9 percent; $p = 0.001$ for trend). Middle SES group women had intermediate values. Black women, compared to White women, were less likely to have *carcinoma-in-situ* or cancer confined to the breast and more likely to have *remote* disease ($p = 0.007$ for trend). The differences in cancer stage by SES group were still present when cases were stratified by race ($p = 0.03$ for White women, $p = 0.04$ for Black women). There were no important differences between

TABLE 1—Percentage of Breast Cancers in Different Stages, Connecticut 1975–85

		Cancers of Known Stage				
Year	Incidence*	N	Ca-in-Situ	Local	Regional	Remote
1975	12.2	1756	3.9	50.7	37.3	8.1
1976	11.3	1641	3.3	52.4	35.6	8.6
1977	11.0	1651	3.4	52.5	35.2	8.9
1978	11.0	1641	3.0	50.7	35.2	11.1
1979	11.4	1716	4.0	51.2	38.2	6.6
1980	11.4	1761	4.0	49.9	38.5	7.6
1981	12.0	1867	3.7	48.5	38.7	9.1
1982	11.6	1814	4.9	49.1	38.5	7.5
1983	12.3	1973	4.5	50.4	37.1	7.9
1984	13.1	2125	6.3	53.1	33.1	7.4
1985	13.6	2220	7.4	53.9	31.5	7.2
Total		20,165				

*Cases per 10,000 women, including those of unknown stage, age-adjusted to 1980 Connecticut population.

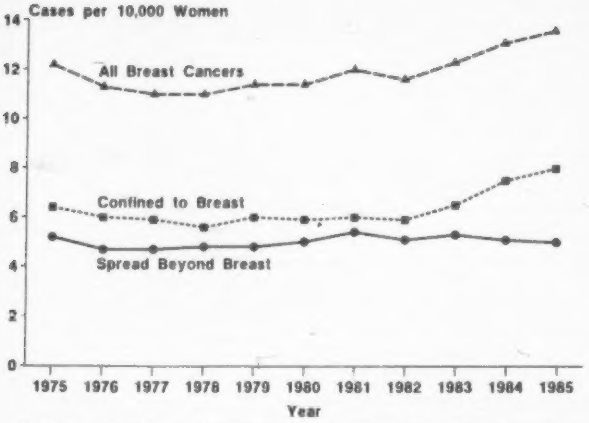

FIGURE 1—Incidence of Breast Cancer by Stage at Diagnosis, Connecticut, 1975–85

any of these subgroups in the percentage of cancers for which the stage was unknown.

The 778 women without census tract information were similar to women in known census tracts in the percentage that were Black or urban residents. Among the 684 of these women for whom the stage was known, the distribution of cancer stage was similar to that of women in known census tracts.

Population-based rates of the different cancer stages were calculated for each SES group. Lower SES women had lower crude and age-adjusted rates of breast cancer overall and of early-stage disease, but a slightly higher rate of metastatic disease (Table 3).

The numbers of cancer cases in each stage were then used to calculate projected breast cancer death rates for the different SES groups (Figure 2). These rates indicate the proportion of women annually diagnosed with fatal breast cancer ("fatal" meaning expected to die from breast cancer within five years from the time of diagnosis). To estimate the expected benefit of a moderately successful early detection program, these death rates were then separated into "preventable" and "nonpreventable" death rates as described. There were small differences among SES groups in the rates of "nonpreventable" deaths. However, lower SES women, compared to higher SES women, had a 25 percent higher projected breast cancer death rate (24.6 vs 19.7 per 100,000) and a greater percentage of those deaths classified as "preventable" (22 percent vs 11 percent). The preventable breast cancer death rate for lower SES women was 2.5 times as great as that for higher SES women (5.3 vs 2.1 per 100,000). A similar analysis projecting 10 years from the time of diagnosis produced parallel results; lower SES women had a 34 percent higher projected death rate (32.0 vs 23.8 per 100,000), and a greater percentage of those deaths "preventable" (20 percent vs 9 percent).

Discussion

The Connecticut Tumor Registry has for many years been an invaluable source of data on cancer incidence trends.[6] Recent advances in our understanding of cancer prevention allow us increasingly to use tumor registries like this as surveillance systems to aid in the design and evaluation of cancer control activities. As surveillance systems, tumor registries not only provide accessible information about the overall success or failure of control activities over time, but also identify subgroups of the population at unusually high risk of fatal disease.

Our analysis of the tumor registry data shows a shift toward early breast cancer detection in Connecticut in the 1980s, as indicated by an increase in the percentage of tumors diagnosed in early stages and a slight decrease in the incidence of later-stage disease. This shift represents an acceleration of the more gradual long-term trend toward earlier diagnosis of breast cancer present since the 1940s.[12,13] It is probable that this long-term trend is responsible (together with an improvement in stage-specific survival rates) for the stabilization of breast cancer mortality rates, in spite of a real increase in breast cancer incidence.[12–14]

No data are available regarding the frequency of screening mammograms or breast examinations in Connecticut. If breast cancer screening is responsible for the shift toward early-stage diagnosis, then we would hope to soon see a further decrease in the population-based incidence of *regional* and *remote* stage disease. Statewide surveillance of breast cancer should therefore periodically assess the incidence of disease by stage to monitor the success of early detection programs.

Both White and Black women in lower SES census tracts were more likely than race-matched women in higher SES census tracts to have later-stage disease at the time of breast cancer diagnosis. A definition of SES based on census tract information subjects the analysis to potential bias caused by an "ecologic fallacy." However, we would expect that the misclassification of women by this group attribute would tend to bias the data toward finding no differences by SES. In addition, our data show a consistent trend over three SES groups, are similar to the analysis of stage by race, and are similar to the findings of stage versus SES found by other authors.[7,8,15,16]

The relation between lower SES and late-stage diagnosis found by others has been noted in the process of correcting for the effect of stage on long-term survival rates. Rather than correcting for tumor stage, we used it as the outcome to measure the success of control activities. This allowed us to assess the effect of early detection programs independent of the effects of primary prevention and treatment programs.

Breast cancer screening programs may detect some early tumors which do not have the potential to progress to clinical cancer. Classifying these tumors as breast cancer would artificially increase both breast cancer incidence and the percentage of tumors diagnosed in early stages. This phenomenon may be partly responsible for the temporal trend and socioeconomic differences in early cancer rates. However, such benign tumors would not alter the population-based incidence of *regional* or *remote* cancer and would not have a significant impact on our projected breast cancer mortality rates.

Although lower SES women are more likely to present with later stage-disease and have poorer stage-adjusted 5-year survival rates[7,9] than higher SES women, they have a lower overall incidence of breast cancer than higher SES women. Therefore it is not immediately apparent which group should be a target of control programs. However, we estimate that these SES differences will lead to a 25 percent increase in deaths for lower SES women compared to higher SES women, with these deaths more than twice as likely to be preventable by early tumor detection.

We have had to make several assumptions in calculating these projected death rates. In particular, we have assumed

TABLE 2—Percentage of Breast Cancers in Different Stages by Socioeconomic Group and Race, Connecticut, 1984–85

Race	SES Group	Cancers of Known Stage				
		N	Ca-in-situ	Local	Regional	Remote
White	Lower	796	6.8	51.8	32.9	8.4
	Middle	1747	6.7	53.1	32.7	7.4
	Higher	897	8.2	54.0	31.9	5.9
Black	Lower	104	3*	44	40	13
	Middle	57	5*	58	26	12
	Higher	13	23*	62	8*	8*
All Women	Lower	907	6.2	50.7	34.2	8.9
	Middle	1822	6.6	53.1	32.6	7.7
	Higher	920	8.5	54.2	31.4	5.9
Total, known SES		3649	7.0	52.8	32.7	7.5

*Represents fewer than five cases.

TABLE 3—Crude (age-adjusted*) Incidence of Breast Cancer per 10,000 Women in Different Stages, 1984–85, by Socioeconomic Group

SES Group	Carcinoma-in-situ	Local	Regional	Remote	Total
Lower	0.65(0.65)	5.33(5.14)	3.60(3.51)	0.94(0.90)	10.8(10.4)
Middle	0.75(0.74)	5.98(5.88)	3.67(3.61)	0.87(0.85)	11.6(11.4)
Upper	1.06(1.04)	6.75(7.29)	3.91(4.08)	0.73(0.80)	12.7(13.5)

*1980 Connecticut population standard.

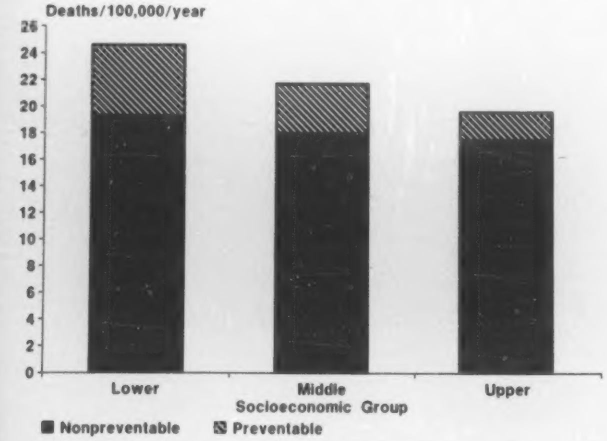

FIGURE 2—Projected Breast Cancer Death Rates for Cases Diagnosed in Connecticut during 1984–85

that within each SES group, long-term stage-specific cancer survival rates would remain unchanged after an early-detection program. This assumption is related to the effect of lead-time on cancer survival and mortality. A theoretical problem exists in assessing the benefit of screening programs if women who have cancers detected early do not have the expected improvements in long-term survival—if these women's deaths are neither prevented nor postponed, but simply occur further from diagnosis. However, over time, as cancers in Connecticut have been detected earlier, long-term survival rates for early-stage disease have actually increased (10-year rates are presently 99 percent for carcinoma-in-situ, 83 percent for cancer localized to the breast). Furthermore, two studies have demonstrated that screening has a beneficial long-term effect on mortality independent of its effect on diagnostic lead-time.[2,3,5] Therefore it is likely that the assumption of constant stage-specific survival rates is sufficiently valid to provide useful estimates regarding the benefit of early detection programs.

While the magnitude of our death rate projections depends on the numerical assumptions we have made regarding disease stage and survival, the trend toward a substantial increase in preventable deaths in lower SES women was present regardless of such assumptions. We believe these projections, combined with the established benefit of early tumor detection in decreasing cancer mortality,[2–5] provide strong justification for prevention programs to direct special attention to lower SES women.

It is probable that women of lower SES present with later-stage disease because of a combination of decreased access to medical care and a decreased awareness of or belief in the importance of early cancer detection. In the National Health Interview Survey, Black women had lower rates of utilization of breast examinations or mammography than White women, and among those >40 years old, were twice as likely as White women to have never heard of mammography.[17] Our data suggest that this difference may be due more to socioeconomic status than race. Data from neighboring Rhode Island indicate that among all women who had not had a mammogram in the past year, the most important reason for not having had the test was that they had believed it was unnecessary.[18] It is important to better define the precise reasons why women in lower SES groups appear less likely to use breast cancer screening techniques.

Connecticut has recently passed a law ensuring that all private and public medical insurance plans—including Medicare—pay for the cost of screening mammography. The Connecticut Department of Health Services is currently working with the American Cancer Society in the state to find ways of better reaching lower SES women. We hope these efforts will improve early cancer detection and thereby contribute to the prevention of breast cancer morbidity and mortality.

APPENDIX

Estimated 5-Year Survival Rates by Cancer Stage

For each SES group, the number of projected breast cancer deaths (D) was calculated as the sum for each race of the following:

$$D = \sum_s \sum_a L_a \times C_{a,s} - \sum_s P_s \sum_a L_a \times C_{a,s}$$
$$= \sum_s (1 - P_s) \times (\sum_a L_a \times C_{a,s})$$

where s = stage, a = age-group, L_a = estimated 5-year actuarial survival for each age-group, P_s = estimated 5-year relative survival for each stage in that SES group (see below), and $C_{a,s}$ = number of actual cases of breast cancer in each age-group and stage. "Nonpreventable" deaths were calculated using the same equation except that the number of actual cases was replaced by the goal number of breast cancer cases in each stage.

The estimated 5-year relative survival rates were generated by adjustment of data from the Surveillance, Epidemiology, and End Results (SEER) program[1] with estimates of previously noted effects of SES on survival rates.[7-9] The rates used were as follows:

	5-year Relative Survival by Cancer Stage				
SES Group	In situ	Local	Regional	Remote	All stages
High	.993	.912	.748	.190	.811
Middle	.989	.899	.685	.174	.782
Low	.986	.886	.622	.158	.754

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Manual Being Developed to Assist Local Boards of Health in Policy-Making

To help improve the capabilities of local boards of health, a manual is being designed by the Association of North Carolina Boards of Health (ANCBH), under a $5,000.00 grant from the W. K. Kellogg Foundation. That financial support will enable ANCBH to review available information on public health policy-making and develop new resources specifically for boards of health in North Carolina, according to Vaughn Upshaw, Administrative Director of ANCBH in Pittsboro, NC. However, he added that the manual would be available to *all* boards of health in the US.

The manual is expected to offer a review of the state statutes in North Carolina, and also provide guidelines for board members to improve their assessment of community needs, policy-making skills, knowledge of agency operations, and advocacy for public health.

Local boards of health are empowered by the state to protect and promote public health. According to ANCBH President Ronald Tucker, "Growing concern for our environment and personal health increases the need for informed local officials." Few resources are available locally to boards of health on how to act effectively, he added. "Board members, health directors, health department staff, and the public will benefit from more knowledgeable and effective boards of health."

For further information, contact Vaughn Upshaw, MPH, ANCBH, PO Box 1009, Pittsboro, NC 27312. Tel: 919/542-4244.

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Sentinel Health Event Surveillance: Skin Cancer of the Scrotum in New York State

AURA L. WEINSTEIN, MPH, HOLLY L. HOWE, PHD, AND WILLIAM S. BURNETT, MD, DRPH

Abstract: Skin cancer of the scrotum is a disease that has been identified as a sentinel health event (occupational) (SHE(O)) that is necessarily occupationally related. The present paper examines the feasibility of using this disease in active cancer surveillance in New York State. After consulting cancer case reports, hospital records, death certificates, and city directories, we obtained occupational data for 17 of 22 cases of nonmelanoma skin cancer of the scrotum diagnosed between 1979 and 1984. Only three cases had occupations previously linked to scrotal cancer, while 11 of 19 cases had one or more possibly contributory medical conditions. The probable underreporting of scrotal cancer cases and incompleteness of occupational data may limit the usefulness of scrotal cancer as a sentinel health event (occupational) unless additional steps are taken. (*Am J Public Health* 1989; 79:1513–1515.)

Introduction

Cancer of the scrotum, which has been associated with exposures to numerous hydrocarbon products,[1] has been identified as a sentinel health event (occupational) (SHE(O)).[2] A SHE(O) is defined as:

> an unnecessary disease, disability, or untimely death which is occupationally related and whose occurrence may: 1) provide the impetus for epidemiologic or industrial hygiene studies, or 2) serve as a warning signal that materials substitution, engineering control, personal protection, or medical care may be required.[2]

SHE(O)s can be broken down into two broad categories: those which are necessarily occupationally related and would be unlikely to occur in the absence of occupational exposures, and those which may or may not be occupationally related. In the original SHE(O) paper,[2] scrotal cancer was identified as one of the three malignant neoplasm SHE(O)s that are necessarily occupationally related.

The Cancer Surveillance Program of the New York State Department of Health is assessing the feasibility of active surveillance using data from the State Cancer Registry to monitor SHE(O) cancers. This feasibility study has examined in detail the occurrence of necessarily occupationally related SHE(O) cancers in New York State and the information on individuals with these cancers obtainable from routine data sources. This paper presents the results of this examination for skin cancer of the scrotum.

Methods

The source of data was the New York State Cancer Registry, which receives reports from physicians, laboratories, and hospitals on all cases of cancer diagnosed in the state, as required by law. Cases are also obtained from death certificates, and through reciprocal interstate reporting agreements. Based on various quality control studies, cancer reporting to the Registry is estimated to be over 95 percent complete for the state.[3] At the time the Registry case listing for this analysis was obtained, case reporting was complete through 1984.

Since it is the epithelial cancers of the scrotum that have been identified with occupational exposures, analysis was confined to these morphologic types. Reporting to the Cancer Registry of nonmelanoma skin cancers of the scrotum, which account for the majority of epithelial scrotal cancers, began in 1979. The present analysis includes cases reported from 1979 through 1984.

Cancer Registry data were supplemented by an examination of hospital records for the cases of nonmelanoma skin cancer of the scrotum identified through the Registry. Hospital records were reviewed for information on smoking history, occupational exposures, course of illness, and any prior or concurrent medical conditions that may have been related to the development of scrotal cancer. Information on occupation was also sought from city directories and from death certificates when appropriate.

Incidence rates for scrotal cancer were computed as average annual rates for the period 1979 through 1984. Rates were age-adjusted to the 1960 United States population to allow for the comparison of rates in different populations.

Results

The New York State Cancer Registry identified 30 incident cases of epithelial skin cancer of the scrotum in New York State residents between 1979 and 1984. This corresponds to an average annual age-adjusted incidence of 0.56 per million males. The epithelial cancers included 22 cases of nonmelanoma skin cancer, all of which were the squamous cell type, and eight other epithelial types such as adenocarcinoma and unspecified carcinoma.

Occupational History

The cancer case report requests information on employer, industry and usual occupation, but only eight of 22 nonmelanoma skin cancer cases had an occupation listed on the case report. These occupations tended to be in the managerial and professional, or service categories. Hospital records, obtained for 19 of the 22, confirmed any occupations listed on the cancer case reports and provided occupations for five additional cases—all in the skilled, semi-skilled, or unskilled labor category. Death certificates supplied occupational information for three more cases, and a city directory provided occupation information for another case. After consulting these sources, five occupations remained unknown. These results are summarized in Table 1.

From the Bureau of Cancer Epidemiology, New York State Department of Health, Albany. Address reprint requests to Aura L. Weinstein, MPH, Research Scientist, Cancer Surveillance Program, New York State Department of Health, Corning Tower, Rm 565, Albany, NY 12237. Dr. Howe is Chief, Division of Epidemiologic Studies for the Illinois Department of Public Health, Springfield. This paper, submitted to the Journal March 22, 1988, was revised and accepted for publication May 9, 1989.

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TABLE 1—Occupational Categories of Males Diagnosed with Nonmelanoma Skin Cancer of the Scrotum, New York State, 1979–84, by Source of Occupational Information

Occupational Category[1]	Cancer Case Report	Hospital Records	Death Certificate	City Directory	Total
Managerial or professional	4	0	0	1	5
Skilled, semi-skilled or unskilled workers[2]	1	5	1	0	7
Service occupations	2	0	0	0	2
Technical, sales and support	1	0	1	0	2
Farm occupations	0	0	1	0	1
Total	8	5	3	1	17

NOTES: 1. Occupational categories derived from summary groupings found in the *1980 Census of Population: Alphabetical Index of Industries and Occupations*, US Department of Commerce, 1982.

2. Includes US Census categories Precision Production, Craft and Repair Occupations, and Operators, Fabricators and Laborers.

Five additional occupations remained unknown.

Three of the persons classified as skilled, semi-skilled, or unskilled workers had occupations that could have put them at elevated risk of scrotal cancer. These included a bridge painter who had reported occupational exposures to graphite and kerosene, a machinist who may have been exposed to carcinogenic cutting oils, and a mechanic for a heating equipment business who had possible exposure to soots and tars. Each of these substances has been linked with scrotal cancer.[1,4] None of the other occupations presented any apparent opportunities for relevant exposures to occur.

Medical Risk Factors

Medical records showed that eight of the 19 squamous cell skin cancer cases with available medical records had a prior history of cancer. Five of these prior cancers were skin cancers (two squamous cell, one malignant melanoma, one unspecified skin, and one mycosis fungoides). One other person had a history of psoriasis, which was treated with coal tar. The patient with mycosis fungoides had undergone PUVA (psoralen-ultraviolet-light) treatments, a regimen often used to treat psoriasis and linked to squamous cell cancers.[5] Other conditions which may have had significance in the development of scrotal cancer are a prior injury to the urinary tract, undescended testis, and "very poor hygiene." The latter condition has been identified as a risk factor for scrotal cancer from the days of Percivall Pott,[6,7] up to a recent case series.[8] There were 11 persons in all who may have had one or more possibly contributory medical conditions.

Other Geographic Areas

The age-adjusted rate (0.56 epithelial cases per million man-years) in New York State for 1979–84 compares with age-adjusted rates of 1.32 per million for Connecticut during the 45-year period 1935–79, and 0.97 per million in Connecticut for 1975–79.[5] More recent data from Connecticut* show an age-adjusted rate of 1.28 per million males for all cancers of the scrotum for the period 1978–84. If epithelial cancers still account for 75 percent of all scrotal cancers reported in Connecticut, then the epithelial scrotal cancer rate is roughly 0.95 per million males, nearly twice that observed in New York.

The age-adjusted incidence rate of 0.52 per million males observed for New York City was compared with that for Metropolitan Detroit, a northern industrial urban area with a similar proportion of black males. The Detroit figures for 1979–84** give an average annual age-adjusted rate of 1.14 cases per million males, which is again about twice that for New York City.

In New York, none of the 30 epithelial cancers of the scrotum reported through 1984 was the (less serious) basal cell carcinoma. Nine of 71 confirmed epithelial cancers in Connecticut from 1935 through 1979 were basal cell, and three of 16 epithelial cancers in Metropolitan Detroit from 1979 through 1986 were basal.

Discussion

Incidence rates of epithelial cancers of the scrotum in New York State and New York City are approximately half those of the Connecticut and Detroit SEER‡ areas, respectively. This may indicate some degree of underreporting, but the extent to which this deficit may also reflect true differences in occupational exposures is not known. The predominance of the squamous cell type also supports the possibility of differential underreporting of the less serious cases.

Skin cancer of the scrotum has been characterized as a cancer whose occurrence is unlikely in the absence of occupational exposures.[2] In the present study, evidence of a possible occupational origin was found for only three of the 17 cases for whom occupational data were available. In contrast, 11 of the 19 persons for whom hospital records were available had prior or co-existing medical conditions that might be related to their scrotal cancers.

One possible interpretation of these findings is that occupational cancer of the scrotum in New York State is at such a low level that "background" or nonoccupational cases predominate. However, the possibility of significant underreporting, particularly of uncomplicated cases, makes it conceivable that an occupationally related spatial or temporal clustering may not be reflected in Registry data.

A related issue is that of the completeness and quality of the occupational data that are available. Less than half of cancer case reports had occupation listed, and those occupations that were listed tended to be of the type where unusual exposures generally do not occur. For the remainder of cases, it was necessary to consult several sources of varying accessibility and completeness. Hospital records provided occupational information for more patients than the cancer case reports, but had to be sent for specially, a process often taking several weeks. New York State death certificates are generally good sources of occupational information, but their usefulness is limited for nonfatal diseases such as skin cancer of the scrotum. It was also not feasible to obtain death certificates for New York City residents, who made up 40 percent of the cases. City directories can provide occupational information, but are not a consistent source since complete listings are not available for all areas. After consulting all these sources, nearly one fourth of the occupations were still unknown. Furthermore, the occupations that were ascertained represent the usual or the most recent occupation; other pertinent occupations may not be recorded.

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*Flannery JT: Chief, Connecticut Tumor Registry, personal communication, 1988.

**Schwartz A: Michigan Cancer Foundation, personal communication, 1988.

‡SEER: Surveillance, Epidemiology, and End Results, the population-based cancer surveillance system established by the National Cancer Institute in 1970.

Steps may be taken to improve the representativeness and completeness of data on scrotal cancer cases and their occupations collected in the future. One would be to remind all physicians of their duty to report nonmelanoma skin cancers of specified sites, and of the proper format and reportable items. A second step might be to send a reminder to all hospital tumor registrars regarding the importance of completing the occupation section on the cancer case report. Obtaining more complete occupational histories would require contacting cases or their next-of-kin directly, often requiring more extensive resources than generally available for surveillance activities.

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The Homeless Families Program—A National RWJ/HUD Initiative

A multi-million dollar public/private program for homeless families has been announced as a cooperative national initiative of the Robert Wood Johnson Foundation and the US Department of Housing and Urban Development (HUD).

The Homeless Families Program is intended to help communities develop the infrastructure necessary to strengthen the potential of homeless parents and their children to function successfully. The Johnson Foundation will make as many as eight two-year grants of up to $300,000 each to eligible cities (population 250,000 or more) or the counties in which these cities are located; Public Housing Authorities (PHAs) in jurisdictions selected under the competition will also be eligible for a share of the Section 8 certificates HUD will set aside for this purpose. The grants and housing certificates will support community-wide projects aimed at providing comprehensive health and supportive services and housing assistance to homeless families suffering from complex, often chronic, problems, including health and mental health problems, inadequate education or job training, and lack of day care and other services.

The goal of the Homeless Families Program is to show that even in the most dysfunctional families, appropriately designed health and support services, combined with suitable housing, can help parents become healthy and hopeful, and avoid becoming permanent members of the nation's underclass. The program also seeks to promote the long-term expansion of permanent housing options for these families.

Interested applicants should send a letter of interest to: **Dr. James O'Connell, Director, Homeless Families Program, Massachusetts General Hospital, 32 Fruit Street, Boston, MA 02114.** Limit the letter to two typed, double-spaced pages. Identify the parties to be involved in developing the application, documenting local government support, and generally outlining the project to be proposed. A contact person should be identified to serve as principal liaison during the application process. Application materials will be sent upon receipt of the letter. **The deadline for receipt of applications is December 22, 1989.** Grant recipients will be announced May 1990. The Robert Wood Johnson Foundation is at College Road, PO Box 2316, Princeton, NJ 08543-2316.

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Carcinogenicity of Dark Liquor

Kenneth J. Rothman, DrPH, Cristina I. Cann, and Marvin P. Fried, MD

Abstract: To investigate whether the non-alcohol content of distilled alcoholic beverages affects the carcinogenicity of the beverage, we conducted an epidemiologic study of laryngeal and hypopharyngeal cancer. We interviewed 384 cases (or spouses, for deceased cases), and compared their responses with those of 876 controls. We classed distilled liquors as dark or light, a rough division according to content of potentially carcinogenic compounds in the beverages. The relative effect on hypopharyngeal cancer risk was much stronger for those who reported high consumption of dark liquor (relative risk = 4.4, 90% confidence interval = 2.9, 6.8) than for those reporting comparable consumption of light liquor (relative risk = 1.3, 90% CI = 0.8, 2.1). For laryngeal cancer, consumption of dark liquor had a smaller effect, and there was little distinction between the effects of dark and light liquor. The data appear consistent with the theory that the non-alcoholic content of distilled alcoholic beverages is a determinant of cancer risk, and that alcoholic beverages act topically rather than systemically in their carcinogenic action. (*Am J Public Health* 1989; 79:1516–1520.)

Introduction

Alcohol consumption is carcinogenic to most of the tissues in the upper aerodigestive tract, including the larynx, pharynx, and mouth.[1-6] Little is known, however, about the mechanism for alcohol carcinogenicity. The effect of alcohol on cancer risk appears greater for tobacco smokers than for non-smokers,[7] and some have questioned whether alcohol consumption is carcinogenic among nonsmokers.[8] Theories about the mechanism of alcohol carcinogenesis include the possibility that alcohol has a topical action, acting as a solvent for carcinogenic compounds, possibly enhancing their penetration through mucosa[8,9]; that alcohol consumption alters the nutritional balance and consequently leads to reduced consumption of dietary preventives that act through a central mechanism on epithelial cells[8,10,11]; and that alcohol directly affects metabolism in a way that increases the susceptibility of certain mucosal cells to cancer.[8] If alcohol acts as a solvent, its carcinogenic effect should be greater among smokers than among non-smokers. In addition, alcoholic beverages that come with already dissolved carcinogens should be more carcinogenic than alcoholic beverages free of such ingredients. This latter effect should be more easily measured among non-smokers, since smokers may inhale enough carcinogens in smoke to overwhelm the possibly smaller effect of carcinogenic substances in the beverages themselves.

Various carcinogens or potential carcinogens have been detected in alcoholic beverages. Beer, for example, has been reported to contain nitrosodimethylamine.[12] Scotch whisky is made by exposing sprouted barley to the smoke of burning peat,[13,14] and many spirits, including bourbon, dark rum, and rye are aged in charred oak barrels.[14] Cognac is aged in limousin oak barrels from which the beverage acquires a high tannin content.[14] Tannin is reportedly carcinogenic in both rats and mice following subcutaneous injection.[15] Despite the presence of these carcinogens in alcoholic beverages, it is uncertain whether the type of alcoholic beverage is an important determinant of cancer risk. For this investigation, we restricted our attention to distilled alcoholic beverages, which have the highest concentrations of alcohol, and evaluated the comparative carcinogenic effects of dark liquor versus light liquor. We classed all whiskeys, dark rum, and cognac as dark liquors, and vodka, gin, and light rum as light liquors. Dark liquors as a group contain much higher concentrations of organic compounds that include higher alcohols, esters, and aldehydes and other congeners.[16] Light liquors are highly distilled alcoholic beverages that are comparatively free of additional chemicals. Many vodkas, for example, are nearly pure ethanol solutions.[16]

Methods

The study population comprised all residents of the Boston Standard Metropolitan Statistical Area during the period January 1, 1982 through December 31, 1984. We prospectively identified cases of laryngeal and hypopharyngeal cancer that were diagnosed in the study population during the study period with the cooperation of 119 otolaryngologists in the area. We would have liked to include cases of mouth cancer in the study, but the otolaryngologists who were our major source for case ascertainment did not see in their practices a high enough proportion of the mouth cancer cases occurring in Greater Boston, whereas they saw the great majority of cases of laryngeal and hypopharyngeal cancer. Before the study, we wrote to all otolaryngologists and head and neck surgeons in Greater Boston and surrounding communities (as far as Worcester); during the study period we continued to approach new practitioners for their cooperation. In all we asked 214 otolaryngologists and head and neck surgeons to cooperate, of whom 119 agreed. Most of the physicians who did not cooperate, some of whom were retired or semi-retired, saw few cancer patients, so that the proportion of potentially eligible cases who were ascertained through physicians' practices is far greater than the proportion of cooperating physicians. We contacted all cooperating physicians monthly to learn of any newly diagnosed eligible cases. All cases had to have a histologically confirmed diagnosis of cancer of the larynx or hypopharynx. We sought written permission from the responsible physician to contact each eligible case.

As an independent source of case ascertainment, we included all cases of laryngeal and hypopharyngeal cancer recorded by the Massachusetts Cancer Registry. The Massachusetts Cancer Registry is a population-based registry to which all hospitals and clinics are required to report all cancer cases, excluding certain skin carcinomas. (At the time that we began this study, the Registry was new and was not an

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adequate source by itself for case ascertainment.) For each case identified by the Registry, we approached the responsible physician and obtained written permission to contact the patient, if the patient had not been identified previously.

For each case identified, we randomly selected as control subjects up to three people from the listing of residents of the same town as the case. These listings are published by each town under state law; with them we were able to ascertain conveniently a geographically representative series of controls that had roughly the same age and sex distribution as the cases. The process called for randomly identifying a place in the town book, and from that place identifying the next resident of the same sex born in either the same calendar year as the case subject or one year earlier or later. This step was repeated for each control subject. The matching criterion for year of birth was relaxed slightly for cases 70 years of age or over. To be eligible, controls had to be alive and still residing in the same town as the case at the time that we attempted to contact them. Many of the potential control subjects that we identified, upon investigation, were not alive; in these instances we continued to pick names from the town listing until we reached a maximum of three controls per case. (Toward the end of data collection, to avoid prolonging the study, we did not seek a full set of three controls for each case.)

All cases and controls received an introductory letter that stated in general terms that we were conducting a health study and desired to arrange an interview. The subjects were then contacted, usually by telephone, to arrange a personal interview conducted by trained study personnel. Those subjects who agreed were interviewed at home using a completely structured interview form. The interviewers were not told that the specific focus of the study concerned alcoholic beverages. The interview covered demographic data, medical history, occupational history, tobacco intake, certain dietary factors, drugs, mouthwashes and gargles, and other items. We obtained detailed data about timing, intensity, and type of exposure to alcoholic beverages and tobacco products. Nearly all interviews were conducted in English; three were in Spanish. For cases who had died or were unable to communicate owing to recent laryngeal surgery, interviews were conducted with the help of a spouse or with the spouse directly. One interview was conducted with the mother of a case, with whom the subject lived all his life except during military service.

For the stratified analyses, the estimation of relative risk (RR) across several strata was performed using maximum likelihood techniques to estimate the common odds ratio.[17]

Results

We identified 679 eligible cases, of whom 425 were ascertained directly from the cooperating physicians, and 254 were ascertained later through the Massachusetts Cancer Registry. We attempted to contact 625 of these cases, and could locate 604. No contact was attempted for 29 because the responsible physician was not cooperating with the study; for 20 because the physician's office requested that we not include the patient in the study; and for five cases, ascertained through the Veterans Administration, because there was no response to several requests from the responsible physician for consent by the patient to include him or her in the study. Of the 604 cases located, 42 were too ill for us to interview. An additional 128 patients had died before we contacted them. Of these 128, we obtained an interview with the spouses of 27, but 71 left no surviving spouse, 12 spouses were not available or did not respond, 10 spouses refused to be interviewed, and eight cases were identified through the Veterans Administration, which did not permit us to contact the surviving spouse. An additional 77 cases refused to participate, eight because of language problems. The total number of cases or spouses who were interviewed was 384; 18 of these interviews were with spouses of living cases who could not communicate readily, and one was with the mother of a case. The "response rate" among cases may be calculated in various ways. Of the 679 originally identified cases, only 57 percent are included among those interviewed, but this group represents 82 percent of those for whom an interview was possible (604 located cases minus 42 who were too ill, 83 who died with no spouse to contact, eight for whom we were not allowed to contact the spouse, and eight with language difficulties).

We identified 2,312 potential control subjects. We received no response to our initial letter addressed to 721 of these people. Many of these, however, no longer lived at the listed address when we attempted contact, or had died. The total, therefore, includes the names of an uncertain number of subjects who were not actually eligible for inclusion as a control. We were able to locate 1,591 of the potential controls; of these, 27 did not participate because of language problems, and 588 did not participate for various other reasons. We interviewed 883 controls, of whom seven gave interviews that we considered unreliable and did not use. The proportion of located controls whom we interviewed (55 percent) is lower than we would have liked, and reflects the increasing difficulty of obtaining cooperation for general population studies.[18]

The most important risk factors to consider as potential confounding factors in a study of laryngeal and hypopharyngeal cancer are age, sex, alcohol consumption, and tobacco use. Total alcohol consumption may be regarded as a possible confounder of the non-alcoholic component of dark or light distilled liquor consumption. Tobacco use may not only confound, but also modify an alcohol effect. Occupational risk factors for these cancers have also been reported, but these risk factors affect only small proportions of the population, and should not be strongly related to type of alcoholic beverage consumed, so that confounding from occupational risk factors was not a concern in this analysis. Socioeconomic status is not a strong risk factor conditional on total alcohol consumption and tobacco use.

We first examined the relation between cancer occurrence and the maximum amount of either dark or light liquor consumed weekly at any time. For analysis, we categorized subjects into three levels of maximum consumption (one drink per week or less, two-six drinks per week, and seven or more drinks per week), and compared those in the highest category with those in the lowest. The crude RR for drinking seven or more drinks per week of light liquor, relative to one or less, was estimated to be 1.7, with a 90 percent confidence interval (CI) of 1.3, 2.3. Controlling sex and age by stratification and calculating the maximum likelihood estimate of a common odds ratio indicated that despite the matching (which can introduce confounding) there was no confounding by these factors. We defined tobacco use in terms of pack-years of cigarette smoking (one pack-year equals 7,305 cigarettes smoked), or pack-year equivalents, based on comparable amounts of tobacco consumed, for cigar, pipe and cigarillo smokers and users of smokeless tobacco. We considered one cigar the equivalent of five cigarettes; one pipeful, one plug, or one cigarillo the equivalent of two cigarettes. For these analyses we divided total tobacco use into two categories, those who reported less than 25 pack-years ("light" smokers) and those who had reported at least 25 pack-years ("heavy" smokers). Finer stratification of smoking produced intolerably small numbers within strata, and the consistently heavy smoking of laryngeal cancer cases precluded using a category boundary less than 25 pack-years. This stratification diminished the point estimate for light liquor consumption slightly, to 1.5. We then controlled simultaneously for tobacco and for the total amount of alcohol consumed; we estimated the total lifetime consumption of ethanol, including beer, wine, and other sources, and we then categorized the total into three levels: fewer than 1,000 drinks, 1,000–14,999 drinks and 15,000 or more drinks. One drink was equivalent to one shot (1½ ounces) of distilled liquor, 12 ounces of beer, and five ounces of wine. Control of tobacco and total alcohol consumption reduced the effect of light liquor consumption, comparing the highest level with the lowest, to an estimated RR of 1.1 with a 90% CI 0.8, 1.5.

The crude RR of consuming seven or more drinks per week of dark liquor, relative to one drink per week or less, was estimated to be 2.6, 90% CI 2.0, 3.2. This value was essentially unchanged after controlling for age and sex by stratification. Controlling for pack-years of tobacco use, however, reduced the point estimate to 1.9, 90% CI 1.5, 2.4. Controlling additionally for total lifetime alcohol consumption reduced the effect estimate to a RR of 1.3, 90% CI 1.0, 1.7.

The difference in effect estimates for the maximum rate of consumption of dark versus light liquor is relatively modest; nevertheless, even after control of confounding by tobacco and total alcohol consumption, there was some indication of a possible carcinogenic effect for dark liquor that did not exist for light liquor.

Cases were classified according to site of tumor into three major categories, hypopharyngeal (84 cases), supraglottic (125) and glottic (175), and the above analyses were repeated for each anatomic site separately. There were no cases of subglottic cancer among the 384 cases interviewed. These results are summarized in Table 1. The effects of dark liquor and light liquor appear similar for the supraglottis and glottis, but dark liquor appears to increase the risk of hypopharyngeal cancer whereas light liquor does not.

To determine whether the effect of dark liquor varied according to level of tobacco use, we examined the effects of drinking various quantities of light and dark liquor separately for light and heavy tobacco users. The distribution of cases for each type of cancer, and controls, is given by the maximum weekly intake of dark liquor and by category of tobacco use in Table 2. There is an increasing pattern of risk for greater maximum weekly consumption of dark liquor for both tobacco-use categories. The trend is again concentrated among hypopharyngeal cases (although it is unstable owing to the small number of cases among those with fewer than 25 pack-years of tobacco use). The joint effect of tobacco use and heavy consumption of dark liquor appears substantially greater than additive for the hypopharyngeal cases. This observation is based on a comparison of the highest versus lowest category of liquor consumption for each of the tobacco categories: the difference is 16.7 in the high-use category and 5.0 in the low-use category. The small number of cases in the latter category affects this comparison also, making the evidence for interaction in these data tentative.

We then conducted multivariate logistic analyses to examine the effects of dark and light liquor consumption in the same analysis. Conditional logistic models, which control for the matching variables of town, age and sex, gave results similar to the corresponding unconditional logistic models,[17] and are not reported. These analyses provided the efficiency to evaluate three levels of consumption of dark and light liquor simultaneously. We categorized subjects according to total number of drinks of dark or light liquor consumed during the subject's lifetime, which we calculated from the detailed histories that we obtained. We used categories of fewer than 200, 200–4,999, and 5,000 or more drinks; those who consumed fewer than 200 drinks formed the referent category. Although the total number of drinks is a function of age, age was found not be confounding, nor was sex, and these variables were therefore omitted from the regression models presented here. The model in Table 3, with terms for two levels of dark and light liquor consumption (in addition to the implied referent category), indicates an association with cancer risk of heavy consumption of dark liquor, and a smaller association for heavy consumption of light liquor. The comparable model restricted to hypopharyngeal cases (Table 4) shows a much stronger effect of heavy consumption of dark liquor and no material association for light liquor consumption. When terms for total alcohol consumption are added to the models in Tables 3 and 4, the effect of heavy consumption of dark liquor is smaller, but some effect

TABLE 1—Relative Risk Estimates for Consuming Seven or More Drinks per Week of Light and Dark Liquor, Relative to Consuming One Drink or Less, with 90% CI, for Three Anatomic Sites, Controlling for Tobacco Use and Total Alcohol Consumption

	Light Liquor		Dark Liquor	
Anatomic Site	RR	90% CI	RR	90% CI
Hypopharynx	1.0	0.6, 1.7	1.6	1.0, 2.5
Supraglottis	1.2	0.7, 1.8	1.2	0.8, 1.8
Glottis	1.0	0.7, 1.6	1.2	0.8, 1.8
All Sites Combined	1.1	0.8, 1.5	1.3	1.0, 1.7

TABLE 2—Distribution of Subjects by Maximum Weekly Consumption of Dark Liquor and by Lifetime Tobacco Use

	Tobacco Consumption					
	Fewer than 25 pack-yrs			25 or more pack-yrs		
	Dark Liquor (drinks/week)			Dark Liquor (drinks/week)		
Subjects	<1	2–6	7+	<1	2–6	7+
Controls	253	53	28	298	88	156
Hypopharynx Cases	3	0	2	25	10	44
Supraglottis Cases	1	2	1	61	16	44
Glottis Cases	20	4	1	68	22	60
RR (all cases combined)	1.0	1.2[a]	1.5[a]	5.4[a] 1.0	5.8[a] 1.1[b]	10.0[a] 1.8[b]
RR (hypopharynx cases)	1.0	0.0[a]	6.0[a]	7.1[a] 1.0	9.6[a] 1.4[b]	23.8[a] 3.4[b]
RR (larynx cases)	1.0	1.4[a]	0.9[a]	5.2[a] 1.0	5.2[a] 1.0[b]	8.0[a] 1.5[b]

[a] Risk ratio with the low-tobacco, low-alcohol category as the referent.
[b] Risk ratio with the high-tobacco, low alcohol category as the referent.

TABLE 3—Relative Risk Estimates and 90% CI for Dark and Light Liquor Consumption in a Multiple Logistic Regression Analysis

Variables	Coefficient	Standard Error	RR Estimate	90% CI
Dark Liquor (200–4,999 drinks)	0.343	0.193	1.41	1.03, 1.94
Dark Liquor (5,000+ drinks)	0.854	0.141	2.35	1.86, 2.96
Light Liquor (200–4,999 drinks)	−0.680	0.234	0.51	0.34, 0.74
Light Liquor (5,000+ drinks)	0.462	0.174	1.59	1.19, 2.11

remains, whereas there is no effect of heavy consumption of light liquor conditional on total ethanol consumption. Table 5 gives the model with total alcohol consumption for hypopharyngeal cases.

We examined the influence of dark liquor consumption for various time periods before the cancer was diagnosed on hypopharyngeal cancer risk. These analyses indicated that consumption of dark liquor within the 10 years preceding diagnosis was much less strongly related to occurrence than was consumption in earlier decades.

Discussion

Methodologically the greatest concern in this study is the response rate, especially that of controls. Only 55 percent of the located controls were interviewed, and an unknown number of eligible controls were not located, in part because many of our subjects came from neighborhoods with high population turnover. The concern is that the subjects actually interviewed could have had exposure histories that differ from those not interviewed, possibly because the exposure is directly or indirectly related to the sequence of steps leading to inclusion in the study. Were the study focusing on total alcohol consumption, this concern would be intensified, since it is easy to image that the amount of alcohol consumed could affect the likelihood of participation. For the exposure of interest, however, which is the type of distilled alcoholic beverage consumed, we conjecture that selection bias from non-participation is a less serious problem. An important bias would have to involve marked selective participation among controls for consumers of dark liquor relative to light liquor or vice versa, independently of the total amount of alcohol consumed. Although there may be social class differences among liquor types that might introduce an association between beverage preference and the decision to participate in the study, the magnitude of these associations would have to be very strong to introduce a worrisome degree of bias into the comparisons reported here.

TABLE 4—Relative Risk Estimates and 90% CI for Dark and Light Liquor Consumption in a Multiple Logistic Regression Analysis (Hypopharyngeal Cases Only)

Variables	Coefficient	Standard Error	RR Estimate	90% CI
Dark Liquor (200–4,999 drinks)	0.396	0.407	1.49	0.76, 2.90
Dark Liquor (5,000+ drinks)	1.483	0.2631	4.40	2.85, 6.78
Light Liquor (200–4,999 drinks)	−0.385	0.405	0.68	0.35, 1.32
Light Liquor (5,000+ drinks)	0.237	0.311	1.27	0.76, 2.12

TABLE 5—Relative Risk Estimates and 90% CI for Dark and Light Liquor Consumption in a Multiple Logistic Regression Analysis Controlling Alcohol Consumption from All Sources (Hypopharyngeal Cases Only)

Variables	Coefficient	Standard Error	RR Estimate	90% CI
Dark Liquor (200–4,999 drinks)	0.019	0.422	1.02	0.51, 2.04
Dark Liquor (5,000+ drinks)	0.559	0.284	1.75	1.10, 2.79
Light Liquor (200–4,999 drinks)	−0.559	0.405	0.57	0.29, 1.11
Light Liquor (5,000+ drinks)	−0.136	0.308	0.87	0.53, 1.45
Total Alcohol (1,000–14,999 drinks)	1.522	0.671	4.58	1.52, 13.8
Total Alcohol (15,000+ drinks)	2.635	0.632	13.9	4.93, 39.4

Another potential concern is the use of surrogate interviews for 12 percent of cases. Cases who were deceased were included if a spouse could be interviewed. We also interviewed the spouses of 18 cases who had difficulty communicating. Spouse interviews presumably vary in accuracy depending on the information sought. The recent history of tobacco use, alcohol use, and other factors of interest is likely to be more accurate than early use. Alcohol consumption, which sometimes occurs without complete spousal awareness, may be particularly susceptible to error in spousal interviews. Here again our focus on type of beverage more than on amount mitigates the potential bias.

We used no surrogates for controls, since controls had to be alive to be eligible. Technically, since controls were matched individually to cases, controls who were alive at the time that the case was diagnosed should be eligible, regardless of whether they died before we attempted to contact them. Our decision not to include such individuals was motivated by convenience; it presents little problem if we can assume that there is no important departure from a dynamic equilibrium in the prevalence of type of distilled alcoholic beverage consumed.

Because all the exposure information in this study was reported by the subjects, there is presumably a substantial amount of misclassification. Some of the misclassification stems from faulty recall, and some from simplifying assumptions that we could not avoid in constructing summary measures of exposure. To the extent that such misclassification is comparable for cases and controls, the effect estimates in this analysis are underestimates of the actual effects.

The carcinogenic effect of alcoholic beverages is already well established.[19–21] When controlling for total alcohol consumption, which permits the evaluation of the effect of the non-alcohol component of alcoholic beverages, the additional cancer risk estimated for heavy consumption of dark liquor is modest.

What is important to note in these findings, however, is the divergent pattern of risks for upper airway cancer experienced by heavy consumers of dark liquor and heavy consumers of light liquor among hypopharyngeal cases. The site localization and the differences in effects between light and dark liquor are predicted by the theory that the carcinogenic action of alcohol is mainly topical, rather than systemic, and that the role of alcohol is to act as a solvent for other carcinogens, increasing the penetration of these carcinogens into epithelial tissue. The interactive effect with tobacco use that was observed is also consistent with this theory. These findings, on the other hand, are difficult to reconcile with alternative theories of alcohol carcinogenesis that suggest that ethanol increases carcinogenic risk through central rather than topical mechanisms.[8]

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Prevention of Childhood Injuries: Evaluation of the Statewide Childhood Injury Prevention Program (SCIPP)

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Abstract: We evaluated the effectiveness of a community-based injury prevention program designed to reduce the incidence of burns, falls in the home, motor vehicle occupant injuries, and poisonings and suffocations among children ages 0–5 years. Between September 1980 and June 1982, we implemented five injury prevention projects concurrently in nine Massachusetts cities and town; five sites, matched on selected demographic characteristics, were control communities. An estimated 42 percent of households with children ages 0–5 years were exposed to one or more of the interventions over the two-year period in the nine communities. Participation in safety programs increased three-fold in the intervention communities and two-fold in the control communities.

Safety knowledge and practices increased in both intervention and control communities. Households that reported participatory exposure to the interventions had higher safety knowledge and behavior scores than those that received other community exposure or no exposure to intervention activities.

We found a distinct reduction in motor vehicle occupant injuries among children ages 0–5 years in the intervention compared with control communities, associated with participatory exposure of about 55 percent of households with children ages 0–5 years. We have no evidence that the coordinated intervention programs reduced the other target injuries—although exposure to prevention messages was associated with safety behaviors for burns and poisonings. (*Am J Public Health* 1989; 79:1521–1527).

Introduction

Injuries are the leading cause of death and a major cause of serious morbidity and disability among children and youth.[1] Yet, there have been relatively few efforts to carry out controlled community prevention trials. Schlesinger, *et al*, evaluated a health education program for families with children less than 7 years of age in a study community using a matched community as a control, but found no reduction in medically attended injuries.[2] The other population-based intervention trials reported have focused on the prevention of specific injury types, such as burns[3,4] and pedestrian injuries.[5,6] We evaluated the effectiveness of the Statewide Childhood Injury Prevention Program (SCIPP)—a community-based injury prevention program designed to reduce the incidence of burns, falls in the home, motor vehicle occupant injuries, poisonings, and suffocations among children ages 0–5 years. Our rationale was that by combining the safety interventions available at the time we could reduce childhood injury rates.

Methods

Study Design

The investigation involved concurrent implementation of five injury prevention projects in nine Massachusetts cities and towns with a total population of 139,810 persons and selection of five control communities with a population of 146,866, matched on demographic characteristics to the intervention communities. The injury prevention projects commenced in September 1980 and continued for a 22-month period, through June 1982. A surveillance system to collect data on incident cases of childhood injuries was established in September 1979, one year before the intervention began, and was maintained throughout the study period to August 1982.[7] Telephone surveys of attitudes, knowledge, self-reported safety practices, and exposure to injury prevention projects were completed before the beginning of the interventions in August 1980 and again after their completion in August 1982.

Intervention Methods

Five intervention programs were selected:

- injury counseling for the parents of young children by pediatricians using the developmentally based Framingham Safety Surveys[8];
- school and community burn prevention education using the Project Burn Prevention curricula[4];
- household injury hazard identification and control through systematic home safety inspections by specially trained local board of health staff[9];
- community-wide promotion of the Massachusetts Poison Control System's telephone information service and public education about poison prevention[10,11]; and
- promotion of child automobile restraint use, particularly for children in preschools and infants leaving maternity hospitals.

The primary target injuries, target age groups, and components for each project appear in Appendix A. The principal focus was families with children ages 0–5 years. Implementation of the projects occurred at health and educational institutions that already served children and their families in the study communities. The five prevention programs were integrated in the same study communities to reinforce each other's preventive messages.

Site Selection

The site selection process involved comparison of data on all 351 cities and towns in Massachusetts for: population

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size and density, age composition, education level, family income, housing characteristics, hospital usage patterns, and characteristics of local pediatricians, school districts, and boards of health,[12–16] and informal information supplied by an advisory committee. The sites were grouped into two comparable sets of intervention and control communities. Details have been published previously.[7]

Measurements

The unit of intervention of this study was the community. Therefore, we evaluated the program's effects on the level of safety knowledge and practices and on the incidence of childhood injuries in the study and control communities with counts of project participants, pre- and post-intervention telephone surveys, and a hospital-based injury surveillance system.

Injury Prevention Project Activities: Reports from each intervention project provided counts of the numbers of persons and households reached and materials distributed. We also conducted a post-intervention telephone survey in August 1982 to estimate exposure to interventions over the preceding two years.

Telephone Surveys: We conducted telephone surveys in both intervention and control communities with use of a random digit dial methodology[17]: pre-intervention in August 1980 (1200 respondents) and post-intervention in August 1982 (914 respondents). The primary criterion for selection into the sample was that at least one child 19 years old or younger lived in the household. The surveys included 95 items that assessed a household's exposure to an intervention (e.g., home visited by a housing inspector), and self-reported safety behavior and knowledge.

In addition, prevention behaviors were measured. We grouped responses to behavior-related questions on the survey to form an index of prevention behavior for each of three injury causes: poisonings (14 items), burns (12 items), and falls (6 items). A committee of injury prevention professionals assigned weights to items within each index and adjusted the weights to produce a scale score of 0–100 points[18] (Appendix B). We used the safety scores as outcome measures. A question concerning the use of auto passenger restraints by children was analyzed as a separate variable.

We used parental responses to questions about exposure to each injury prevention intervention to classify households into one of three hierarchical levels: 1) no exposure; 2) community exposure, i.e., the family received general injury prevention information, such as pamphlets, from a community source; or 3) participatory exposure, i.e., the family received specific intervention materials, direct person-to-person counseling, or modifications of a household hazard from at least one SCIPP-funded or related project. We classified households by their greatest degree of exposure to any one intervention. Participatory exposures could have been provided by our own projects or those of other community agencies.

Injury Surveillance: The sampling plan and operational characteristics of the injury surveillance system have been described elsewhere.[7] Briefly, we defined cases as children and adolescents (ages 0–19 years) who resided in one of the 14 communities and who had an injury that required medical treatment at an emergency room, admission to a hospital, or resulted in death. We recorded injury cases at 23 hospitals that provided care for an estimated 93 percent of all pediatric injuries in the population.

The five target injuries for intervention efforts were motor vehicle occupants; poisonings due to medicinals and household products and plants; burns; falls from stairs, ladders, other building structures and furniture; and suffocation from objects.

Statistical Analysis

Changes in Knowledge and Practices: We assessed differences between pre- and post-intervention telephone survey responses for the intervention and control communities by means of the safety scores for burns, poisonings, and falls in the home and the responses to the motor vehicle restraint behavior question. Since the two surveys consisted of different respondents, we conducted a two-factor analysis of covariance (ANCOVA) with socioeconomic status (SES) as the covariate, using the SAS PC computer statistical package.[19] Green's SES score constituted the measure of SES in this analysis.[20] The two factors were the community effect (control vs intervention sites) and the time effect (pre- vs post-intervention). We construed an interaction effect between the two main effects as evidence of differential patterns of safety by community type and time of measurement. A statistically significant interaction would need further investigation with use of post hoc comparisons to determine whether the pattern of interaction revealed an impact of the prevention programs in the study communities.

Effects of Exposure on Knowledge and Practices: We used the nature of exposure (no exposure, community exposure, and participatory exposure) as the main factor in a one-factor ANCOVA with SES as the covariate; the safety indices for poison, burns, and falls in the home were the dependent variables. For motor vehicle occupant behavior, we evaluated the association between nature of exposure (exposed or not exposed) and response to the survey question about child use of auto restraints by chi-square and logistic regression that adjusted for SES.

Changes in Injury Incidence: We compared age-specific injury rates in the pre-intervention period (approximately one year) to the during-intervention period (approximately 22 months) in the control and intervention communities. The post-intervention period was too brief to analyze. We computed Cochran-Mantel-Haenszel (CMH)[21] statistics to test the association between the rate changes (pre- to during intervention) and community type (control or intervention) with control for SES. The measure of SES used in this analysis was median family income of the community, which we stratified into three levels to achieve groups of roughly similar size: less than \$20,000, \$20,000–25,000, and more than \$25,000. Within each combination of age group and injury type, we established a 2 × 2 contingency table.

Results

Exposure to Interventions

During the 22-month period of intensive intervention in the nine intervention communities, direct counts of participation showed that an estimated 42 percent of households with children ages 0–5 years participated in the activities of the SCIPP prevention projects. Pediatric counseling, which included a strong child auto seat component, reached 21 percent of households with children ages 0–5 years; child auto passenger safety programs reached 17 percent; Project Burn Prevention in the community reached 10 percent; home hazard inspection reached 5 percent; and the poison control system reached 1 percent.

We obtained completed telephone surveys for 1,200 households in the pre-intervention survey and 914 in the post-intervention survey; in all we reached the mothers of 4,351 children ages 0–19 years, about 5 percent of all children in the 14 communities. Success in completion of surveys for eligible telephone numbers varied from 59 percent in the pre-intervention survey, when we made only five call-backs, to 85 percent in the post-survey when we made up to 10 call-backs, but did not differ between intervention and control communities. The refusal rate for eligible households was 9.8 percent in the pre-intervention survey and 6.0 percent in the post-survey. The demographic characteristics of the interviewee samples were similar. For the purposes of this evaluation, we used only those households with children ages 0–5 years.

Table 1 shows that even prior to the beginning of the program, households in both study and control communities were exposed to childhood safety messages by a variety of means. We found no significant differences in pre-intervention safety exposures between intervention and control communities.

Over the course of the intervention, the level of participatory exposure for households with children 0–5 years increased three-fold in the intervention communities and two-fold in the controls. Overall, intervention communities' had a higher percentage of participatory exposure than control communities, 62.4 percent vs 38.2 percent (95% confidence intervals of difference 13.9, 34.5). Similar differences hold for participatory exposure in all five target injuries (Table 1).

Changes in Safety Knowledge and Practices

The SES-adjusted mean safety scores for burns- and falls-related behaviors increased from the pre- to post-intervention survey to the same extent in both the intervention and control communities (Table 2) The poisoning safety index showed some effect of exposure. We found no interaction effects for any of these indices.

Effects of Exposure to Interventions on Knowledge and Practices

We found an association between level of participation and safety behaviors for burns and poisoning but not for falls (Table 2).

For the auto restraint use variable, we evaluated only two exposure groups (exposed and non-exposed). Reported use of child auto restraints was 22.2 percent higher in households that had exposure to prevention programs than in households not so exposed (Table 2). To hold the effects of SES constant, we computed the logistic regression of the probability of reporting restraint use on exposure status and SES. The coefficient for exposure status was 0.47 ($\chi^2 = 31.2$, $p = 0.0001$). With employment of methods commonly used in econometrics to calculate the first derivatives of the logistic regression function,[22] the difference between exposed and non-exposed households in reported child restraint use adjusted for SES reduces to 11.6 percent.

Changes in Injury Incidence

Figure 1 shows the pattern of injury incidence rates for target injuries sustained by children ages 0–5 years in the intervention and control communities over the three-year period. At every point, the incidence rate for intervention communities is lower than that for controls. This difference reflects the difference in SES between the intervention and control communities and disappears with adjustment of the rates for SES.

The crude incidence rates for specific target injuries for children ages 0–5 years appear in Table 3. The overall incidence for all target injuries increased in both intervention and control communities from the pre- to the during-intervention period. In the intervention communities, however, the incidence of motor vehicle occupant injuries decreased 54 percent, while rates rose in the control communities. With control of SES, the odds in favor of a motor vehicle occupant injury in the pre-intervention period versus the during-intervention for the intervention communities was 2.78 times as large as that for the control communities.

Discussion

The principal positive finding of this study is a distinct reduction in motor vehicle occupant injuries among children

TABLE 1—Exposure of Households with Children Ages 0–5 Years to any Injury Prevention Program as Measured by Responses to Pre- and Post-Intervention Telephone Surveys

		Community Type and Time			
		Intervention Communities		Control Communities	
Target Injury	Exposure	Pre-	Post-	Pre-	Post-
		Number			
		230	184	256	157
		Percent			
Motor Vehicle Occupant	Participatory	19.1	55.0	14.1	34.4
	None	80.9	44.9	86.0	65.6
Burns	Participatory	13.0	58.7	13.3	29.3
	Community	10.4	13.8	7.4	22.9
	None	76.5	27.5	79.3	47.8
Falls	Participatory	12.2	54.5	10.2	28.7
	None	87.8	45.5	89.8	71.3
Poisonings	Participatory	13.5	55.6	13.3	32.5
	Community	77.0	41.3	75.8	60.5
	None	9.6	3.6	10.9	7.0
Any Target Injury*	Participatory	20.4	62.4	19.9	38.2
	Community	71.7	34.4	70.7	57.3
	None	7.8	3.2	9.4	4.5

*Unduplicated count of exposure to any one of the intervention programs.

TABLE 2—Changes in Safety Knowledge and Behaviors for Households with Children Ages 0–5 by Community Type and Time and by Level of Exposure to Intervention

Target Knowledge Behaviors	By Community Type and Time				By Level of Exposure in Intervention and Control Combination		
	Intervention Communities		Control Communities				
	Pre-	Post-	Pre-	Post-	Participatory	Community	None
	Percent Using Restraints						
Child auto restraint use[b]	49.1	65.0	49.6	63.3	70.9	—	48.7
	Adjusted Mean Safety Scores[a]						
Burns	44.6	49.2	43.7	46.8	49.6	46.8	43.8
Falls[b]	28.4	30.3	29.7	30.7	31.2	—	29.2
Poisonings	31.9	34.3	27.2	30.5	36.0	29.4	23.8

a) Mean safety score adjusted for SES; possible score is 0–100.
b) Single level of exposure to falls and child auto restraint interventions.

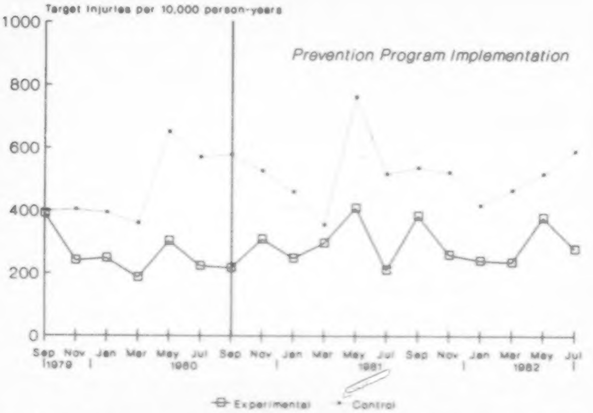

FIGURE 1—Injury Incidence Rates for Target Injuries for Children Ages 0–5 Years in Experimental and Control Communities by 60-Day Periods before and during Injury Prevention Program Implementation

ages 0–5 years in the intervention compared with control communities. This finding is associated with evidence that about 55 percent of households with children ages 0–5 years participated in the interventions. Because many factors may have influenced the use of child auto restraints, however, the observed decrease in motor vehicle occupant injuries cannot be attributed to SCIPP alone. We have no evidence that the coordinated intervention programs reduced the other target injuries—although exposure to prevention messages was associated with safety behaviors for burns and poisonings. These findings suggest three major issues: the design of injury prevention evaluations, the relationship between injury incidence and preventive efforts that alter safety knowledge and behaviors, and the efficacy of efforts to reduce childhood motor vehicle occupant injuries.

The study was designed with the community as the level of intervention and analysis. Because it was impractical to randomize communities, we attempted to match communities on socioeconomic and demographic characteristics with use of the most currently available census data, 1970; injury morbidity rates were unavailable. By 1980, however, the control communities differed demographically from the intervention communities, and had higher proportions of poor and Hispanic households. This demographic change was reflected in higher baseline injury rates in the control communities, a finding which is in itself important. We addressed this problem by controlling for SES in all analyses.

Exposure to injury prevention messages occurred from sources other than our project. The media promoted childhood safety as did the pediatric and public health literature[23–25]; schools introduced safety curricula into their health education programs; the Massachusetts Legislature was debating a child auto restraint bill which, no doubt, led some motivated families to purchase these devices. This exposure of control sites introduced an element of misclassification into the comparison of intervention and control communities. It would have been impossible, however, to have eliminated these sources; estimating the magnitude of their effect on the findings is indeed difficult.

Evaluation of the effect of an intervention depends on the case definition of an injury and the method of ascertainment. We used an emergency room visit as the definition of the least severe injury. It is possible that some of our prevention messages may have altered the level of injury severity and use of the emergency room. For example, knowledge of the use of cold water following a scald burn might have reduced sufficiently the severity of burns so as to avoid hospitalization. Moreover, differential access to treatment by area of residence and socioeconomic status may have influenced ascertainment of cases.

High baseline levels of safety behaviors influenced our ability to show community effects. For example, our pre-intervention survey found that 73 percent and 69 percent of households in study and control communities, respectively, had smoke detectors. At the end of the study period, this level had changed to 82 percent and 84 percent, respectively. Given the low incidence of burns associated with house fires, the study did not have adequate power to evaluate this change. In other cases, the level of exposure to an intervention may have been too low to demonstrate an effect. For example, household inspection and correction of hazards is a highly efficacious intervention,[9] but we reached only 5 percent of all target households in this trial. These design considerations emphasize the importance of using injury morbidity rates by cause at the local level to influence the selection of community sites, target injuries, and intervention strategies.

Finally, the study design did not permit us to disaggregate the changes in knowledge and behavior by the type of intervention strategy. We cannot comment on the relative efficacy of patient counseling by pediatricians, school edu-

TABLE 3—Changes in Target Injury Rates for Children Ages 0–5 Years in Intervention and Control Communities

	Injury Rates[a]				Mantel-Haenszel Odds Ratio[b]	
	Intervention		Control			
Target Injuries	Pre-	During	Pre-	During	OR	95% CI[b]
Motor Vehicle Occupant	46.54	21.54	44.53	60.77	2.78	1.66,4.66
Burns	52.56	59.68	75.01	106.03	1.26	0.84,1.90
Falls	127.07	175.02	252.03	262.44	0.78	0.61,1.00
Poisonings	31.76	36.14	92.71	92.71	0.95	0.57,1.58
Total target[c]	262.80	297.11	477.42	523.96	0.99	0.83,1.19

a) Injury Rates: per 10,000 Children per year using 1980 US census data.
b) Controlling for SES.
c)Total target injuries include a small number of suffocations on objects other than food.

cational curricula, community promotion, or home inspection as the technique to best influence behavior. Different intervention techniques will likely have different effects, and these need further study.

We do know that there were several components to the efforts to reduce motor vehicle occupant injuries. These included promotion by pediatricians of infant and child auto restraint utilization, an in-hospital training program for health professionals and car seat loan programs, establishment of a Child Auto Passenger Safety Resource Center at the state health department with community presentations for parents and providers at preschools and other settings, and statewide advocacy for a mandatory child restraint law which was passed in December 1981. Participation in these activities combined with media attention may have increased safety behavior in the intervention communities as compared to the non-intervention sites. There is good evidence from studies of cardiovascular disease control programs that combined mass media and comprehensive community-based interventions can succeed in improving a community's health status.[26–28]

There is good reason to believe that the prevention technologies promoted through our educational efforts were of differing levels of efficacy. Thus, while we may have exposed our population to all the injury-specific messages equally, we observed a protective effect on auto occupant injuries, primarily, because the child auto restraint device is a highly efficacious technology, and auto crashes occur with sufficient frequency so that one can measure an effect.

Length of exposure to injury prevention efforts may be important to the success of community efforts such as SCIPP. We maintained the interventions and surveillance system for only 22 months because of limited resources. The benefits of changed safety knowledge and practices may manifest themselves more slowly and over a longer time frame. Schelp, in a community-based intervention in Sweden, showed decreases in injury rates after four years of activity.[29] In retrospect, our study was grossly underfunded given its ambitious nature.

Finally, we must acknowledge constraints in implementing "text-book" interventions in a real world setting. We cannot understate the logistics and difficulty of managing a set of complex interventions in several communities simultaneously. Our efforts to persuade local health agencies and community boards to take more of a lead role were thwarted by the unavailability of local morbidity data, a lack of financial incentives, and a lack of media attention to the injury problem. It may be that child auto injury was the only well-recognized injury issue.

In designing our project, we defined childhood injuries broadly and selected prevention methods from those available to us in 1979, when we had little evaluation data available. For example, the results of the evaluation of Project Burn Prevention were not available until 1982, when our intervention was nearly completed.[4] Our target injuries were limited by the availability of these interventions. Were we to redesign the SCIPP intervention today, we would look carefully at all the effective interventions discussed by Rivara[30] and others[31,*] and select only those that have strong evidence of efficacy. Future interventions must include the active participation of local public health agencies, a media component to increase the awareness of both the public and professionals, and evaluation over a longer time period. One must convince these local agencies of the importance of the injury problem, obtain their commitment of resources, and assure that the requisite local morbidity data are available for targeting injuries which are prominent in the community and for evaluation of the interventions.

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APPENDIX A

Injury Prevention Intervention

Intervention	Objectives	Targets	Components
Child Auto Passenger Safety Project	• To increase utilization of infant and child auto restraints • To reduce motor vehicle occupant injuries to children ages 0–5 years.	• Parents of newborns and young children • Health providers with access to maternity patients • Hospitals	• Survey of current hospital practices • In-hospital training of maternity and child birth educators • Resource Center for technical assistance • Promote car seat loan programs • Outreach to preschools • Observational study of restraint usage • Passage of mandatory child restraint law
Home Injury Prevention Project	• To reduce the number of hazards in the home environment • To incorporate injury prevention into traditional role of health agents • To reduce burns, poisonings, falls, and product-related injuries	• Families with children under age 5 years • High-risk housing • Landlords and homeowners	• Systematic enforcement of State Sanitary Code • Safety counseling to hazardous household practices • Distribution and installation of safety devices • Follow-up evaluation
Pediatric Accident Prevention Project	• To provide a developmentally-oriented, focused counseling system for pediatric primary care settings • To increase awareness and safety practices in parents of young children • To reduce burns, poisonings, motor vehicle injuries, falls, choking, and drowning	• Parents of children under age 5 years • Pediatricians • Well-baby clinics • Visiting nurses	• Age appropriate safety surveys • Counseling by health providers • Free safety supplies • Follow-up home assessment/evaluation
Poison Control System	• To promote appropriate treatment of the poisoned patient • To promote awareness and proper storage of poisonous substances	• Parents and older siblings of children under age 5 years • Hospital staff • Pharmacists Other retailers	• Promote emergency consultation care of poisoned patient • Educate health professionals • Promote poison information hotline • Promote availability of Ipecac • Mail surveys and site visits to pharmacies and other retailers • Free distribution of Ipecac displays, phone stickers and pamphlets • Poster contest for grades K–2
Project Burn Prevention	• To institute a burn prevention curriculum in schools • To increase awareness of burn hazards, escape from house fires, and clothing ignition • To reduce all causes of burns	• Parents of young children under age 5 years • School-age (4–18) children enrolled in public and private schools • Community groups	• Train teachers in curriculum use • Free multi-media kits • Train volunteers • Promotion through libraries, fire & police, day care centers, civic organizations and youth groups • Follow-up evaluation

APPENDIX B

Telephone Survey Questions and Weights Used to Calculate Safety Indices for Burns, Poisonings, and Falls

Telephone Survey Questions	Weight
Poison Safety Index	
1. Store drugs/pain relievers on open shelf, in cabinet, in closet, in medical cabinet, or someplace else and the place is shoulder height?	2.6
2. Store drugs/pain relievers on open shelf, in cabinet, in closet, in medical cabinet, or someplace else and the place is locked?	20.8
3. Store drain cleaners on open shelf, in cabinet, in closet, in medical cabinet, or someplace else and the place is shoulder height?	5.2
4. Store drain cleaners on open shelf, in cabinet, in closet, in medical cabinet, or someplace else and the place is locked?	22.0
5. Store drain cleans in the original container?	4.2
6. Store liquid bleach on open shelf, in cabinet, in closet, in medical cabinet, or someplace else and the place is shoulder height?	2.0
7. Store liquid bleach on open shelf, in cabinet, in closet, in medical cabinet, or someplace else and the place is locked?	2.0
8. Never left bleach in a measuring cup?	2.2
9. Use kindergard latches on the cabinets?	2.8
10. The first response to poisoning is call poison control center for advice?	15.4
11. The first response to poisoning is call doctor/nurse for advice?	6.8
12. Have Ipecac Syrup at home?	9.6
13. Have poison phone sticker posted and the number is 232-2120 or 1-800-682-9211?	2.4
14. Have poison phone sticker posted and the number *is other than* 232-2120 or 1-800-682-9211?	2.0
Total:	100.0
Burn Safety Index	
1. None of the cord of the electrical appliance hangs below the counter?	3.5
2. The best thing to put on burn is cool water?	8.6
3. Have a smoke detector in the building?	18.4
4. Have smoke detector installed in the bedroom hallway, or the basement, or all rooms?	14.6
5. Family has fire escape plan?	4.4
6. Family has tried fire escape plan?	5.6
7. Have safety plugs on electric outlets (yes/no question)?	2.0
8. Have safety plugs on *all* the outlets?	2.2
9. Have safety plugs on *some* of the outlets?	2.1
10. Do not have additional heat source at home?	3.7
11. The hot water temperature at home is 125 or below?	32.6
12. The fuses at home do not blow frequently?	2.0
Total:	99.7
Fall Safety Index	
1. Do not have broken window?	3.3
2. There are screens or barriers on window?	7.8
3. Have safety gates at either the top or bottom of the stairs?	26.3
4. Have safety gates at both the top and bottom of the stairs?	45.0
5. All the stairways in the building have railings?	14.3
6. None of the railings are unsteady?	3.3
Total:	100.0

Human Cryptosporidiosis Associated with an Epizootic in Calves

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DAVID A. DARGATZ, DVM, MS, AND JOHN M. CHENEY, DVM, MS

Abstract: An outbreak of human cryptosporidiosis occurred among previously healthy persons in a veterinary medical teaching hospital. Human illness began after admission of calves from a farm which had been experiencing an epizootic of neonatal diarrhea due to *Cryptosporidium*. The clinical syndrome in humans was characterized by watery diarrhea, abdominal cramping, flatulence, and headache. Cryptosporidiosis was confirmed by zinc sulfate flotation of fecal specimens in four persons, three of whom had been responsible for the care and treatment of infected calves. A fourth patient had washed her husband's soiled clothing and appeared to have been infected indirectly through fomite contamination. Among 112 persons surveyed, 26 (23.2 percent) had a diarrheal illness during the outbreak and nine met the case definition of a diarrheal illness lasting at least three days. These persons were more likely to have had contact with a calf with diarrhea than were 52 referents who did not become ill during the outbreak. (*Am J Public Health* 1989; 79:1528–1530.)

Introduction

Cryptosporidium is an intestinal protozoan parasite, recognized relatively recently as a human pathogen,[1] despite its original identification over 70 years earlier.[2] Following the description of the first human case of cryptosporidiosis in 1976, the disease was reported rarely, and primarily in immunocompromised persons.[3] Since 1982, the number of reported cases has increased dramatically.[4] Initially this increase was noted in patients with acquired immunodeficiency syndrome[5] but recent reports indicate that cryptosporidiosis also occurs in immunologically normal persons.[6–8] In immunocompetent persons, cryptosporidiosis is manifested as an acute, self-limiting diarrheal illness lasting five to six days.[4,6–8] Cases of cryptosporidiosis have been described in travelers,[9] residents of day care centers,[10] animal handlers,[8,11] veterinary students,[12–14] and following consumption of contaminated community water supplies[15] or surface waters.[16] A previous description of an outbreak of cryptosporidiosis among five veterinary medical students[13] was ascribed to contact with experimentally infected calves. Recently, infection of at least 10 students who had had contact with 1-to-3-day-old calves in a laboratory setting was described.[14] We report here an outbreak of cryptosporidiosis among previously healthy veterinary students associated with a spontaneous epizootic of bovine cryptosporidiosis in a clinical setting.

Methods

Outbreak in Humans

The index case was a 34-year-old female veterinary student whose duties included nursing care, sample collection, and treatment of calves with diarrhea which were subsequently identified as infected with cryptosporidia. On April 6, 1987, one week after her initial contact with the calves, she complained of watery diarrhea, abdominal cramps, nausea, fever (greater than 99°F), headache, vomiting and flatulence; she was ill for two weeks. She received medical attention and was found to have cryptosporidiosis after parasitologic examination of her stools.

During the next two weeks, several additional students and faculty members complained of gastrointestinal symptoms. A large proportion of these persons were associated with the food animal services. When cryptosporidia were identified in calves hospitalized in the isolation unit, a potential relationship between the animal and human diarrheal illnesses became apparent.

An investigation was initiated on April 17, 1987. A questionnaire distributed to all students and food animal hospital staff included items on recent gastrointestinal illness, and exposure to potential risk factors in the hospital and at home. Dates of potential exposure to calves in the isolation unit were determined from student duty rosters. Persons experiencing diarrheal illness and others with food animal contact were asked to submit stool samples.

Parasitologic examination for cryptosporidia oocysts consisted of fecal flotation by centrifugation of zinc-sulfate (sp. gr. 1.18) to which aqueous iodine had been added.[17] Cover slips from these flotations were examined by light and phase contrast microscopy. Fecal smears were examined after methanol fixation and Ziehl-Nielsen staining.[18] Analysis of risk for specific exposures was accomplished by calculation of odds ratios (OR) for persons experiencing a diarrheal illness, using those who remained clinically normal as the comparison group.[19] Confidence intervals (95% CI) for these risk estimates were obtained by exact testing.[20]

Epizootic in Calves

An outbreak of diarrheal illness in beef calves occurred on a farm in northern Colorado. Virtually all of the 91 calves born after February 1987 developed postnatal diarrhea. Newborn calves continued to develop diarrhea during the first week of life as the calving season progressed. Due to the continuing nature of the diarrheal disorder, four newborn calves with severe diarrhea and dehydration were admitted to the Veterinary Teaching Hospital on March 23, 1987 and placed in the isolation unit. Additional affected calves were admitted weekly until June 6. On April 16, 1987 a fecal flotation performed on one of the inpatient calves revealed the presence of cryptosporidia oocysts. Upon testing of additional inpatient calves, 6/6 (100 percent) were positive for *Cryptosporidium*. Among 20 calves tested which remained on the farm, 17 (85 percent) were positive. Other potential enteric pathogens identified in calves from this farm included

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Campylobacter, *E. coli*, bovine coronavirus, and bovine rotavirus.

Results

Diarrhea occurring between March 16 and April 26, 1987 was reported by 26 of 112 questionnaire respondents (Table 1). With the exception of one unconfirmed illness, all cases were clustered in the three-week period between April 6 and April 26, 1987. In order to attempt to exclude short-lived illness due to viral and bacterial enteric pathogens and dietary upset, a case definition of diarrheal illness lasting at least three days was adopted. Nine respondents (7.4 percent) met this criterion and were defined as cases in subsequent analyses. Cryptosporidiosis was confirmed by identifying oocysts in stool specimens from four persons among six who reported diarrhea. Conversely, cryptosporidia were not detected in any of nine other individuals without a history of recent diarrhea.

Fourth year students had more frequent direct contact with diarrheic calves, adult cattle, and other animals than third year students during the outbreak. Three of the confirmed cases were fourth year students who had responsibility for therapy of the diarrheic calves in the isolation unit between April 1 and the date of diagnosis. The fourth was a female hospital employee whose spouse was a student. She denied contact with calves in the isolation unit. However, her husband had contact with the calves during the weeks of April 6 and 13 and she had washed his fecally contaminated clothing. She became ill during the week of April 13, complaining of a mucoid and watery stool, abdominal cramps, fever, nausea, vomiting, and flatulence.

A watery stool was reported by all patients. Abdominal cramping, flatulence, and headache were predominant features of the clinical disorder. The symptoms reported by persons with parasitologically confirmed cryptosporidiosis were generally similar to those reported by the unconfirmed cases.* The mean duration of illness in confirmed cases was seven days; the illness lasted over two weeks in one person.

The association between personal and occupational risk factors and the occurrence of gastrointestinal illness during the outbreak was examined.* The reference group comprised all fourth year student and staff respondents without a history of gastrointestinal illness (N = 52). Odds ratios of 13.2 and 11.8 were found for contact with a calf with diarrhea and having taken a calf's temperature, while intubating a calf for anesthesia carried no excess risk. The exact confidence intervals around these risk estimates were wide due to the small number of persons affected but did not include one. Contact with dogs, cats, or foals with diarrhea did not increase risk.

No association was found between human gastrointestinal illness during the outbreak and having a child in a day care center, or consumption of untreated surface water. The community is served by a municipal water system which uses complete conventional treatment consisting of chlorination, alum and polyelectrolyte coagulation and flocculation and rapid sand filtration through multimedia filters. Cases were not more likely than members of the comparison group to have had another illness, a history of frequent diarrhea or blood transfusion, or to have taken oral antibiotics during the previous month. A high proportion of both affected and unaffected students claimed to be under physical or psychological stress.

TABLE 1—Frequency of Symptoms, Duration of Illness and Isolation of *Cryptosporidium* during Outbreak of Gastrointestinal Illness in a Veterinary Teaching Hospital, March 16 to April 26, 1987

	Fourth Year Students	Third Year Students	Hospital Personnel	Total
Persons Responding	45	47	20	112
Persons with Diarrhea	10	13	3	26
Persons with Symptoms ≥3 Days	8	0	2	9
Confirmed Cases	3	0	1	4

Discussion

The outbreak of cryptosporidiosis reported here reemphasizes the importance of this organism as a cause of gastrointestinal illness in previously healthy, immunocompetent persons.[6–8,15,16,21,22] Travelers, residents of day care centers, homosexual males, and children in developing countries constitute important risk groups for this cosmopolitan parasite.[23]

In the current outbreak, calves served as the source of human infection. This occurred despite the facts that veterinary students have frequent contact with diarrheic patients of many species, and that *Cryptosporidium* is reported to display little, if any, host specificity.[4] However, it is not clear if *Cryptosporidium* oocysts from animals other than cattle are infectious for humans. Previous reports of cryptosporidiosis in veterinary medical students have also involved contact with calves. In one, a student who provided supportive care of two calves with cryptosporidiosis became ill,[12] and in another, five students caring for experimentally infected newborn calves were affected.[13] In a recent report,[14] *Cryptosporidium* was detected in 10 of 20 students who had contact with 1-to-3-day-old calves in a student laboratory. The incubation period ranged from five to 14 days after exposure and the diarrhea lasted 30 hours to 16 days. Two infected students had only nausea or headache, suggesting that unrecognized infections may occur.

Reports of cryptosporidiosis in research workers[11,24] and animal handlers[8,24] with direct contact with feces from experimentally and naturally infected calves add to the evidence that the young calf is an important source of human infection. The reason for the consistent role of the calf as a source of cryptosporidiosis for humans is not clear, but may be related to the high prevalence of infection among calves,[25] and shedding of large numbers of oocysts for prolonged periods.[26] The preeminence of diarrheic calves as a source of transmission for zoonotic cryptosporidiosis dictates the necessity for exposed persons to adopt stringent hygienic precautions including protective clothing when working in an environment potentially contaminated with *Cryptosporidium*.

Fomites appear to constitute a hazard for the indirect transmission of *Cryptosporidium*. In the current outbreak, one confirmed human case had contact with clothing soiled with fecal material from infected calves as the likely source of infection. The oocysts of *Cryptosporidium* are resistant to desiccation and several disinfectants[27] and are thus likely to persist in the environment. Evidence that waterborne transmission occurs supports this hypothesis.[15,16]

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*Complete data available on request to authors.

The outbreak began approximately two weeks after the first calves were admitted, but the status of shedding of *Cryptosporidium* by specific animals was not determined until after the onset of the epidemic in humans. Instituting hygienic measures and awareness of the existence of an epidemic of gastrointestinal illness among students with contact with calves may have contributed to the cessation of clinical cases after the week of April 20, despite the fact that calves continued to be admitted to the hospital from the farm in question.

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Public Health Service Implementation Plan for Responding to Nursing Shortage

The US Public Health Service has developed an implementation plan in response to the recommendations of the Secretary's Commission on Nursing, according to O. Marie Henry, RN, DrPH, PHS Chief Nurse Officer. Reporting recently to the Advisory Council on Nurses Education of the Health Resources and Services Administration, in Rockville, MD, Dr. Henry indicated that the plan is intended to coordinate activities and resources among PHS agencies and establish a focus within the US Department of Health and Human Services for responding to the nursing shortage.

In general, the plan continues current activities in such areas as research, financial assistance, and program development, within available resources, and also establishes new initiatives. The plan is organized around nine major objectives in the general categories of resource development, maintenance, and utilization, Dr. Henry said.

Copies of the 16-page "Public Health Service Implementation Plan for the Secretary's Commission on Nursing Final Report" are available from the Office of the USPHS Chief Nurse Officer, Room 14A-40, 5600 Fishers Lane, Rockville, MD 20857. Tel: (301) 443-0577.

6T

Polymedicine Use among Community Resident Older Women: How Much of a Problem?

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Abstract: In a community-based study of older women describing the prevalence of specific medication combinations, the risk of deleterious consequence from polymedicine was concentrated in a minority of older women. There were few prevalent medication combinations, and many of the more common combinations were unavoidable. These results suggest that broad, community-based drug education programs may be less effective than those targeting medical providers and multi-drug users. (*Am J Public Health* 1989; 79:1537–1540.)

Introduction

As a group, the elderly suffer a larger burden of illness than younger adults, and consume disproportionately more medication;[1–3] community-based surveys report that about 70 percent of those over 65 years old take at least one medication.[4,5] Estimates of the average number of drugs taken at any one time varies considerably, from about 1.5 drugs to more than four drugs, depending on the age distribution of the sample, and whether those reporting no drug use were included in the average.[4,5] Moreover, age-related physiologic changes may increase the likelihood of adverse drug effects. Hence, older persons are thought to be at increased risk for the deleterious consequences of "polymedicine."[6,7] The size of this risk depends on the prevalence of specific medication combinations, yet little is known about the prevalence of specific medication combinations in the older community population, and none of the available information is specific to women. Women typically use more drugs than their male counterparts, although this association wanes somewhat with age.[8] However, women may be more susceptible than men to adverse drug reactions as they age because they experience larger declines in lean body mass.[9,10]

The purpose of this paper is to describe the prevalence of specific medication combinations in a community resident population of older White women.

Methods

Sampling Design

Study participants included 807 White women aged 65 and older living in a stable, relatively homogeneous urban community in a 20 census tract area in Northeast Baltimore. During 1984, a random sample of 6,540 addresses was enumerated from this community. The enumeration effort had a 95 percent response rate and yielded 1,203 homes occupied by at least one woman over age 65. If a household contained more than one woman over age 65, one was randomly selected to participate. Of the 1,203 eligible subjects, 807 (68 percent) agreed to participate. Details of this survey are available elsewhere.[11]

Prescription Drug Use

We asked respondents if they had taken any prescription medications during the past month and examined the original containers for all medications reported. Name, date of prescription, dose, and reason for taking each medication (using the information on the medication label) were recorded. Bottles were available for 91.6 percent of all prescriptions. Respondents provided information on other cases and, when necessary, dispensing pharmacies were contacted for clarification. Each drug was assigned the appropriate therapeutic drug code, using the American Medical Association's drug classification system.[12] The therapeutic codes were condensed into 10 drug categories. A number of drug categories with very low prevalence in this community dwelling population were omitted from the analysis. These included anti-malarial agents, anti-neoplastic agents, dermatologic compounds, and chelating agents.

Non-Prescription Drug Use

The interviewer also asked respondents if they had taken any non-prescription medications during the week prior to the interview. If the respondent answered affirmatively, the interviewer read the respondent a list of types of non-prescription drugs, including vitamins, laxatives, over-the-counter pain relievers, antacids, sleeping pills, and insulin, asking the respondent which ones she had taken.

Results

The 807 women in this sample report a mean age of 72.7 years, with a range of 65 to 99 years. Nearly 6 percent are over 85 years old. On average the respondents have completed 9.9 years of schooling, although 40 percent had

TABLE 1—Number of Prescription Medications Reported

	Percent of Respondents	
Number of Medications	Prescription	Non-Prescription
0	26.0	23.3
1	21.1	34.0
2	19.8	23.5
3	12.8	12.8
4	9.0	4.4
5	5.2	1.5
6 or more	6.1	0.5

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TABLE 2—Drug Use by Therapeutic Category and Subcategory

Drug Category	Percent Reporting Use
Cardiovascular renal agents	55.8
Digitalis glycosides	11.3
Antiarrhythmic agents	7.0
Antianginal agents	15.0
Agents for peripheral and cerebral vascular disorders	2.5
Antihypertensive agents	19.9
Diuretics	36.4
Blood formation, volume, coagulation agents	2.7
Drugs for deficiency anemias	2.4
Anticoagulants	0.4
Homeostatic, nutrient agents	10.8
Agents for hyperlipidemia	0.4
Vitamins/minerals	1.2
Replenishers and regulators of water, electrolytes and nutrients	9.1
Agents affecting calcium metabolism	0.2
Drugs used for pain relief	19.5
Strong analgesics	2.4
Mild analgesics	3.6
Agents used to treat migraine	0.1
Agents used to treat gout	1.3
Antirheumatic agents	14.0
Central nervous system drugs	13.8
Sedatives and hypnotics	3.8
Antianxiety agents	6.8
Antipsychotic and antimanic agents	0.7
Anticonvulsants	0.9
Antidepressants	3.8
Hormones	14.6
Adrenal corticosteroids	2.3
Estrogens, progestogens	2.4
Anterior pituitary and hypothalamic agents	0.1
Agents for hyperglycemia	5.7
Agents for thyroid disease	4.6
Respiratory/allergic disorder drugs	5.8
Bronchodialators and antiasthmatics	2.4
Nasal decongestants	0.4
Antitussive agents	0.1
Antihistamines	2.7
Cold remedies	0.6
Antimicrobial agents	5.2
Penicillins	1.2
Cephalosporins	0.6
Macrolides, lincomycin and clindamycin	0.6
Tetracyclines	0.5
Sulfonamides	0.6
Nitrofurans	0.5
Antifungal agents	0.4
Drugs used in ophthalmology	3.7
Agents used to treat glaucoma	2.6
Mydriatics and cycloplegics	0.1
Ocular anti-inflammatory agents	0.9
Gastrointestinal agents	8.7
Agents used to treat peptic ulcer	2.2
Antidiarrheal agents	1.5
Laxatives	0.4
Antiemetics	0.2
Miscellaneous	3.7

completed less than eight years of schooling and 10 percent had completed at least some college.

Prescription Drug Use

On average, the women in this sample report using 2.02 prescription drugs each, with a range of 0 to 11 drugs. The distribution of prescription medication use is presented in Table 1. Over one-fourth report using no prescription medications, while nearly 20 percent report using four or more prescription medications. Table 2 breaks down drug use by therapeutic category, and by class within category.

TABLE 3—Percent of Respondents Reporting Use of Each Category of Non-Prescription Drug

Drug Category	Percent Reporting Use
Vitamins	31.5
Laxatives	18.5
Pain relievers	31.0
Antacids	20.0
Sleeping pills	2.4
Insulin	1.2
Others	18.5

About 9 percent of all prescription medications had label instructions to use "as needed," including: 36 percent of the allergy/respiratory disorder drugs, 20 percent of the gastrointestinal agents, 27 percent of the central nervous system agents, and 21 percent of the drugs used for pain.

Use of Non-Prescription Drugs

The mean number of non-prescription drugs used in the week prior to the interview was 1.4 with a range of 0 to 7 (see Table 1). Twenty-three percent of the sample report taking no non-prescription drugs during the week prior to the interview, while 19 percent report taking three or more non-prescription drugs. Table 3 reports the percent of respondents reporting use of each category of non-prescription drug.

Analysis of Drug Combinations

To describe the potential extent of polymedicine in this sample, we ascertained the number of respondents reporting the use of each possible two-drug category combination by cross-tabulating all the drug category variables. The chi-square test with Yates' correction for 2×2 tables was applied to differences between observed and expected values. Tables 4 thru 6 report the results of this analysis. It is important to remember that the number of cases reported for any drug combination is bounded by the prevalence of the least frequently occurring drug in the combination.

Table 4 presents observed and expected frequencies for combinations of two prescription drugs. Of the 45 potential combinations, eight (18 per cent) occurred with significantly greater than expected frequency. It is noteworthy that about one in eight respondents (109/807) report taking the combination of cardiovascular renal agents and drugs for pain. This is not surprising since 44 percent of the women reported having high blood pressure, and 60 percent reported having arthritis, the two most common indications for use of cardiovascular renal agents and pain medications, respectively, in this sample.

Table 5 reports the observed and expected frequency of respondents reporting two-way combinations of one prescription drug and one non-prescription drug. Of the 60 combinations, five (about 8 percent) occurred with greater than expected frequency. Four combinations of cardiovascular-renal agents and non-prescription drugs have prevalence rates in this sample of over 10 percent. More than 25 percent of the sample (219/807) report using both cardiovascular renal agents and over-the-counter pain relievers. The combinations of cardiovascular renal agents with vitamins, laxatives, and antacids each occur in at least 10 percent of the sample.

Table 6 performs an analysis for two drug combinations of non-prescription drugs. Of the 15 potential combinations,

TABLE 4—Observed and Expected ( ) Frequency of Respondents Reporting Each Possible Two-way Combination of Prescription Drug Category

		2	3	4	5	6	7	8	9	10
1. Cardiovascular Renal Agents	Obs.	16	76**	109**	77**	75	29	24	20	47
	(Exp.)	(12)	(49)	(88)	(62)	(66)	(26)	(23)	(17)	(39)
2. Blood Formation Volume, Coagulation Agents	Obs.		2	8	5	2	2	2	1	4
	(Exp.)		(2)	(4)	(3)	(3)	(1)	(1)	(1)	(2)
3. Hemeostatic and Nutrient Agents	Obs.			22	17	16	6	6	3	12
	(Exp.)			(17)	(12)	(13)	(5)	(5)	(3)	(8)
4. Drugs Used for Pain	Obs.				36**	35**	18**	10	8	15
	(Exp.)				(21)	(23)	(9)	(8)	(6)	(14)
5. Central Nervous System Drugs	Obs.					18	9	9	6	24**
	(Exp.)					(16)	(7)	(6)	(4)	(10)
6. Hormones and Agents Affecting Hormonal Mechanisms	Obs.						9	4	7	8
	(Exp.)						(7)	(6)	(4)	(10)
7. Respiratory and Allergic Disorder Drugs	Obs.							8*	3	7
	(Exp.)							(3)	(2)	(4)
8. Anti-microbials	Obs.								0	6
	(Exp.)								(2)	(4)
9. Opthalmologic Agents	Obs.									4
	(Exp.)									(3)
10. Gastrointestinal Agents	Obs.									—
	(Exp.)									—

*p < .05
**p < .01

four (26 percent) occurred with greater than expected frequency. Two combinations of non-prescription drugs occur in more than 10 percent of the sample: vitamins and laxatives, and over-the-counter pain relievers and antacids. The latter was also reported with greater than expected frequency.

Discussion

This analysis indicates that there is a wide range of drug taking patterns among older women living in the community. While half the sample reported taking no or only one prescription medication, 20 percent reported using four or more prescription drugs. The use of non-prescription medications is equally varied. Hence, community dwelling older women are not homogeneous in their "risk" for the potentially deleterious effects of polymedicine.

An analysis of medication combinations demonstrates few prevalent combinations in this sample of community dwellers. In addition, many of the more prevalent combinations may be therapeutically unavoidable. For example, the combination of cardiovascular renal agents and pain relievers (prescription and non-prescription) occurs with high fre-

TABLE 5—Observed and Expected () Frequency of Respondents Reporting Two-way Combinations of Prescription and Non-Prescription Drugs

		Vitamins	Laxatives	OTC-Pain Relievers	Antacids	Sleeping Pills	Insulin
Cardiovascular Renal Agents	Obs.	123	84	219	96	14	9
	(Exp.)	(142)	(83)	(210)	(90)	(11)	(6)
Blood Formation Volume, Coagulation Agents	Obs.	9	6	14	5	0	0
	(Exp.)	(7)	(4)	(10)	(4)	(1)	(0)
Hemeostatic and Nutrient Agents	Obs.	26	22	40	22	4	0
	(Exp.)	(27)	(16)	(41)	(17)	(2)	(1)
Drugs Used for Pain	Obs.	54	42*	71	42	7	1
	(Exp.)	(50)	(29)	(73)	(31)	(4)	(2)
Central Nervous System Drugs	Obs.	47*	24	60	35*	6	3
	(Exp.)	(35)	(20)	(52)	(22)	(3)	(1)
Hormones and Agents Affecting Hormonal Mechanisms	Obs.	40	24	33	20	32*	4
	(Exp.)	(37)	(22)	(37)	(21)	(27)	(3)
Respiratory and Allergic Disorder Drugs	Obs.	15	9	23	8	1	1
	(Exp.)	(15)	(9)	(22)	(9)	(1)	(1)
Anti-microbials	Obs.	13	8	19	11	1	1
	(Exp.)	(13)	(8)	(20)	(8)	(1)	(1)
Opthalmologic Agents	Obs.	10	10	15	6	0	1
	(Exp.)	(9)	(5)	(15)	(6)	(1)	(0)
Gastrointestinal Agents	Obs.	19	16	30	32*	3	0
	(Exp.)	(22)	(13)	(33)	(14)	(2)	(9)

*p < .05
**p < .01

TABLE 6—Observed and Expected ( ) Frequency of Respondents Reporting Two-way Combinations of Non-Prescription Drugs

		Laxatives	OTC-Pain Relievers	Antacids	Sleeping Pills	Insulin
Vitamins	Obs.	59*	131	64	10	3
	(Exp.)	(47)	(119)	(51)	(6)	(3)
Laxatives	Obs.		80	45**	9*	2
	(Exp.)		(69)	(30)	(4)	(2)
OTC Pain Relievers	Obs.			97**	13	6
	(Exp.)			(75)	(9)	(5)
Antacids	Obs.				5	1
	(Exp.)				(4)	(2)
Sleeping Pills	Obs.					1
	(Exp.)					(0)
Insulin	Obs.					—
	(Exp.)					—

*$p < .05$
**$p < .01$

quency simply because many older women suffer from both hypertension/heart disease and arthritis. Other frequently observed combinations of medications result from the need to temper side effects—the co-occurrence of cardiovascular renal agents and hemeostatic/nutrient agents represents the need to add potassium supplements to diuretic therapy. Similarly, the frequent joint occurrence of over-the-counter pain relievers and antacids may reflect self-medication of the gastrointestinal side effects of aspirin.

Some of the more dangerous combinations fortunately occur with relatively low frequency. Some combinations of central nervous system agents and pain medications could be potentially dangerous. Overall, this combination occurs in about 4.5 percent of our subjects. In addition, six persons using a central nervous system agent and seven persons using a pain medication reported self-medicating with non-prescription sleeping pills.

Several caveats are in order. This sample derives from a community-based population of White women. While representative of a sizable portion of the older population, they can be expected to differ in important ways from other elderly. Treatment-based populations, both inpatient and outpatient, would overrepresent high medication users relative to this sample. Men and persons of other races may also differ from White women in their use of medications. Furthermore, these analyses use prevalence data. It is likely that our reports of drug use underestimate the incidence of drugs used for acute conditions.

Because relatively few older women report using many drugs, this analysis suggests that the risk of deleterious effects from polymedicine is not significant for the majority of community resident older women. A large minority, about 20 percent, are however, at substantial risk. Educational interventions directed toward providers which aim at encouraging appropriate prescribing and monitoring practices when treating older adults are already underway in many areas of the country. Given that this analysis suggests that the risk of deleterious effects of polymedicine is concentrated in a minority of older women, educational interventions should be directed at these community-dwelling, high medication users as well.

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Factors Affecting Place of Death of Hospice and Non-Hospice Cancer Patients

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Abstract: We identified factors associated with death at home for 28,828 hospice and non-hospice cancer patients in 13 counties of western Washington State. Hospice participation was found to be the variable most strongly associated with death at home. Admission to hospice appears to override the tendency for certain subgroups of patients, such as the extreme elderly and those diagnosed close to death, to die in an institutional setting. These findings are discussed with respect to the problem of selection bias. (*Am J Public Health* 1989; 79:1549–1551.)

Introduction

Death at home has been associated with availability of hospice programs[1,2] and whether or not the hospice is hospital-based or home care-based.[1,3–5] In addition, variables such as case mix, availability of caregiver support, and federal policies associated with the new prospective payment system and the Medicare hospice benefit are suspected of influencing length of stay in hospitals and hospice[4–14] and may affect place of death. A number of these variables were addressed in this analysis of factors associated with death at home for hospice and non-hospice cancer patients. Hospice participation was predicted to be a major determinant of death at home.

Methods

The study population included all 28,828 residents of 13 Washington State counties who died during 1980–85 with cancer as a primary cause of death, and whose diagnosis was made prior to death. Among this population, 6,762 were admitted to one of the hospices in the area. Cancer diagnosis information was obtained from the Fred Hutchinson Cancer Research Center Cancer Surveillance System and hospice data were obtained through the cooperation of 21 hospices.

We selected two types of independent variables: 1) previously established association with place of death and/or traditional adjustment or controlling variables (age at death, sex, race, marital status, rural/urban nature of county of residence, stage and site of cancer at diagnosis, presence of a contributory cause of death on the death certificate, time from diagnosis to death, and year of death); 2) policy-related variables (type of ownership of the hospital involved in continuing care of the patient, whether or not the hospital was affiliated with or had its own hospice program, whether or not the hospital was affiliated with or had its own home care program, and type of hospice program utilized).

The effect of independent variables on the relation between hospice participation and death at home was measured by a statistic derived from the relative risk.[15] In each stratum of a variable, the "scaled" relative risk was calculated as the stratum-specific relative risk for home death divided by the overall relative risk pooled across all strata. A variable that does not modify the hospice effect on place of death will have a scaled relative risk of 1.0 for all strata.

Logistic regression analyses were based on 26,500 patients with known values for all relevant variables. The dependent variable was coded as dying at home versus dying elsewhere. Independent variables in the logistic regression were modeled as dummy variables.[16]

Results

Hospice patients were 2.8 times as likely to die at home as non-hospice patients, the strongest association with place of death of any variable studied. Patients in hospice Home Health Based 1 and 2 programs were more likely to die at home than other hospice patients (Table 1). Inpatient hospice participants had a frequency of home death that was closer to the frequency for non-hospice than for hospice patients. The association of place of death with hospice participation yielded a p value $<.0005$ whether hospice was categorized as

TABLE 1—Place of Death, among Cancer Patients, by Type of Hospice or Non-Hospice Care

	Place of Death (percent)				
Type of Care	At Home	Hospital	Nursing Home	Other	N
Non-Hospice	16.3	63.6	19.1	1.0	22066
Hospice*	45.3	42.3	10.8	1.6	6762
HH-Based 1	53.2	36.8	8.2	1.8	3794
HH-Based 2	41.3	45.7	11.6	1.2	1642
Inpatient	25.1	56.7	16.7	1.6	1013
Community-Based/Volunteer Intensive	35.8	44.1	18.6	1.3	313
Total N					28828

*HHB1: General home and hospice home care delivered by a Medicare Certified Home Health Agency.

HHB2: Hospice home care only, delivered by a Medicare Certified Home Health Agency.

Inpatient: Hospice care delivered in a hospital setting.

Community-Based/Volunteer Intensive: Hospice care delivered by organizations supported by donations and volunteer services.

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TABLE 2—Percent of Hospice and Non-Hospice Patients Dying at Home; Relative Risk and Scaled Relative Risk for Dying at Home Associated with Hospice By Patient Demographic and Medical Variables (N = 28,828)

Variables	Hospice % (N)	Non-Hospice % (N)	RR	Scaled RR
Age at Death (years)				
0–19	69.2 (26)	25.9 (189)	2.7	1.0
20–64	44.3 (2488)	17.9 (7682)	2.5	.9
65–74	45.0 (2397)	16.4 (6580)	2.7	1.0
75–84	46.0 (1494)	15.4 (5064)	2.7	1.1
85+	49.2 (356)	12.0 (2351)	4.1	1.5
Unknown (N = 1)				
Marital Status				
Single	37.9 (264)	14.2 (1405)	2.7	1.0
Married	47.6 (4400)	18.2 (12694)	2.6	1.0
Divorced	36.4 (623)	13.7 (2159)	2.7	1.0
Widowed	43.4 (1470)	13.6 (5778)	3.2	1.2
Unknown (N = 35)				
Residence				
Rural	39.7 (398)	20.1 (2326)	2.0	.7
Semi-Urban	55.0 (762)	18.7 (3939)	2.9	1.0
Urban	44.4 (5602)	15.1 (15801)	2.9	1.0
Unknown (N=0)				
Time from Diagnosis to Death				
<30 Days	50.0 (70)	5.1 (1228)	9.8	3.6
>30 Days	45.1 (6598)	17.0 (19970)	2.7	1.0
Unknown (962)				
Year of Death				
1980–82	40.4 (2142)	15.4 (11567)	2.6	1.0
1983–85	47.5 (4620)	17.3 (10499)	2.7	1.0
Unknown (N = 0)				
Hospital Ownership				
Local Govt	44.3 (784)	16.4 (3188)	2.7	1.0
Non-Profit	44.8 (5080)	16.8 (14691)	2.7	1.0
For-Profit	45.8 (341)	11.5 (1017)	4.0	1.4
Federal Govt	42.0 (150)	11.3 (1294)	3.7	1.4
Unknown (2283)				

dichotomous (yes/no), as a five-category variable as in Table 1, or when limited to the four categories of hospice.

The variables that had both a statistically significant direct association with place of death (home versus other) and a significant interaction with hospice in affecting place of death are shown in Table 2. All interactions were significant with $p \leq .005$ except for year of death ($p = .02$).

The positive association of hospice with death at home was stronger for the very old (age 85+), for those not living in rural counties, for those diagnosed close to the time of death (the highest scaled relative risk), and for patients using for-profit and federal government hospitals instead of non-profit and local government hospitals. There was only a minor variation in the hospice association with place of death across categories of marital status and year of death.

A logistic regression model for place of death (home versus other) was fit including all variables from Table 2. The interactions of each of the variables with hospice participation (yes/no) were included in exploratory analyses. Initially 13 constructs (all levels of a variable or levels of interaction) were included. Constructs with a p-value of $.05/13 = <.004$ were retained in the model.

The final model is presented in Table 3. The main effects and interactions all had p-values below .0001. The pattern of main effect relative risks closely followed those in Table 2. Among the interactions, only that of hospice with age and with time from diagnosis to death remained significant. As in Table 2, the positive association of hospice with death at home modifies the relation between place of death and the two variables, age at death and time from diagnosis to death. Patients age 85 and older, and those diagnosed close to death were more likely to die at home than would be predicted by age at death, time from diagnosis to death, and hospice alone.

TABLE 3—Relative Risks, Coefficients, and Standard Errors for Death at Home, Linear Logistic Model (n = 26,500)

Variables	Relative Risk	Coefficient	Standard Error
Intercept	–	−1.62	.04
Age at Death (years)			
<65	1.1	0.12	.05
65–74*	1.0	0	–
75–84	1.0	−0.04	.05
85+	0.8	−0.25	.08
Hospice Participation			
Non-Hospice*	1.0	0	–
Home Health-Based 1	5.1	1.63	.06
Home Health-Based 2	3.6	1.28	.07
Inpatient	1.6	0.48	.09
Community-Based	1.9	0.66	.14
Marital Status			
Currently Married*	1.0	0	–
Single	0.7	−0.34	.07
Divorced/Widowed	0.8	−0.27	.04
County Location			
Urban*	1.0	0	–
Semi Urban	1.3	0.24	.04
Rural	1.4	0.32	.05
Time from Diagnosis to Death			
<30 Days	0.3	−1.36	.14
>30 Days*	1.0	0	–
Year of Death			
1980–1982*	1.0	0	–
1983–1985	1.2	0.14	.03
Hospital Ownership			
Non-Profit*	1.0	0	–
Local Government	0.9	−0.07	.04
For-Profit	0.9	−0.09	.08
Federal Government	0.6	−0.46	.08
Interactions			
Hospice × Time from Diagnosis-Death less than 30 days	6.0	1.80	.29
Hospice × Age at Death			
Hospice × <65	0.9	−0.09	.08
Hospice × 75–84	1.1	0.12	.09
Hospice × 85+	1.6	0.49	.14

*Reference Category for Dummy Variables

Discussion

Hospice participation is clearly the variable most strongly related to whether or not a cancer patient will die at home. It appears that once a patient is admitted to a hospice program, the strong philosophy supporting home deaths overrides the likelihood for patients with certain characteristics to die in hospitals or nursing homes.

Because this is an observational study, it is impossible to determine if the strong association of hospice with home death is causal or if it is due to the selection of hospice by people who prefer to die at home. It is likely that at least some of the association is a causal effect of hospice itself.

The relative risk of death at home for hospice patients varies across type of hospice program. These data are consistent with the results of the National Hospice Study[1,3] where patients cared for by hospices not controlling hospital beds were almost twice as likely to die at home. It is still the case that, relative to non-hospice patients, hospice patients have a much greater likelihood of dying at home. Further, the effect of hospice also seems to be to counteract factors such as age that reduce the likelihood of death at home.

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The Impact of Media Coverage of Nancy Reagan's Experience on Breast Cancer Screening

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Abstract: In surveys of random samples of women 50 years of age and older residing in two communities in Long Island, New York, both within-subject (cohort) and independent-sample comparisons were made before and after the media announcements of Nancy Reagan's breast cancer. Knowledge of lifetime risk of breast cancer increased significantly only in the cohort comparison, while self-perception of risk did not increase. Small proportions of women surveyed, however, reportedly were influenced to contact a health professional (6–8 percent) and to have their first mammogram (1.5–2 percent) which they attributed directly to Mrs. Reagan's experience with breast cancer. (*Am J Public Health* 1989; 79:1551–1552.)

Introduction

While the breast cancer experiences of public personalities could have an effect on awareness and use of cancer screening tests, no population-based studies have been published on the response to such media events. Black, *et al.*[1] found no evidence for improvement in stage distribution of breast lesions treated in two hospitals in New York City after the public announcements of breast cancer in the wives of the US President and Vice President in 1974. On October 15, 1987, Nancy Reagan's breast cancer was announced and in October–November newspapers and both local and network television reported that the cancer had been detected at an early stage by mammography. We examined the short-term effect of the news about Nancy Reagan's breast cancer experience on the breast cancer awareness and screening practices of women on Long Island.

Methods

As part of a community-based study,[2] a random sample of 254 female licensed drivers 50 years of age and older residing in two non-adjacent townships on Long Island were mailed a survey in September–October 1987 (time-1), with a $2 token prepayment. This survey included questions about past screening behavior (mammography, breast physicals, and self-examination), concerns about mammography, knowledge of breast cancer risk, and demographic characteristics. After three mailings (including certified mail) the final response rate to the time-1 survey was 68 percent (N = 183), excluding those who had died or moved out of the area. The 173 who had responded prior to the announcement of Mrs. Reagan's surgery in the media were sent a follow-up survey in November–December (time-2). The 142 women responding to both surveys comprise the cohort group. Surveys were also sent in November-December to an independent sample of women randomly selected from the same two Long Island communities (time-2), with a response rate of 71 percent (N = 205). To assess the changes in responses from time-1 to time-2, chi-square was used for the independent samples and a matched-pair or McNemar chi-square test was used for the cohort.

Results

There were no significant differences between these

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groups of women in age, education, or income (Table 1). The follow-up surveys at time-2 directly asked women whether they had heard about Nancy Reagan's experience with breast cancer and to what extent their screening behavior had been influenced by that experience. Some 85–88 percent of the women surveyed at time-2 had been exposed to the news of her breast cancer in the month after the event through both newspapers and TV coverage, and the majority of women (57–62 percent) also heard about it on the radio. About 8 percent of the cohort respondents and 6 percent of the independent sample (time-2) respondents reportedly contacted a doctor or other health professional as a result of Mrs. Reagan's experience; none contacted either the American Cancer Society or a hospital.

In the period between the two surveys, 17 of the 142 women in the cohort had mammograms, three of whom had never had a mammogram previously. Among these 17 women, 12 said that their decision was not influenced "at all" by Nancy Reagan's experience, three were "somewhat" influenced, and only one was influenced "a great deal." The latter four included the three women (or 2.1 percent of the 142) who had their first mammogram in this period.

Among the independent sample time-2 respondents, 17 women had a mammogram in the October to December period; five of these women had never had a mammogram previously. Of these 17 women, three who said that their decision was influenced by Mrs. Reagan's experience had never had a mammogram previously (3 of 205 or 1.5 percent).

Changes in proportions of correct responses to knowledge questions were slight but in the direction of improved knowledge. In the cohort at time-2, the proportion of correct responses to the question regarding lifetime risk of breast cancer had increased significantly (Table 2) but there was no increase in the proportion of cohort respondents who considered it "very likely" or "somewhat likely" that they would get breast cancer.

Discussion

This study examined the population-level response to the media attention paid to a public figure's experience with breast cancer. Almost all women over the age of 50 had heard about Nancy Reagan's experience through the media and a very small proportion of the defined population sample was influenced in terms of a behavioral change—i.e., seeing a health professional or having their first mammogram. The slight increase in knowledge of risk was not accompanied by an increased sense of personal susceptibility to breast cancer.

Data from the 1987 Behavioral Risk Factor Surveillance System (BRFSS) analyzed by the Centers for Disease Control[3] showed an absolute increase of 12 percent in screening mammography use between the first two months of 1987 and the last two months of 1987 among women who had a medical checkup in that year. The CDC noted the temporal association with Mrs. Reagan's breast cancer but could not attribute the increase in mammography to this event because the effects of the media coverage of the Nancy Reagan news could not be distinguished from the media attention paid to other cancer control promotions in the same year.

TABLE 1—Selected Characteristics of Cohort and Independent Samples

Characteristics	Cohort (N = 142)	Independent Samples Time-1 (N = 183)	Independent Samples Time-2 (N = 205)
Mean Age (years)	62	63	63
Education Level Completed			
≤High school	63 (45.3)	82 (45.8)	99 (48.8)
1–3 years of college	37 (26.6)	47 (26.3)	57 (28.1)
College graduate +	39 (28.1)	50 (27.9)	47 (23.1)
Annual family income			
≤$15,000	27 (20.3)	39 (24.2)	45 (24.1)
$15,000+	106 (79.7)	122 (75.8)	142 (75.9)

TABLE 2—Percent Changes in Response from Time-1 to Time-2

Variables	Cohort	Independent Samples
Correct answer to what proportion of women will get breast cancer at some time in their lives	+9.2%[a] (p = .049)	+3.5% (p = .412)
Perceives self at risk for getting breast cancer	−5.6%[b] (p = .136)	−1.6% (p = .711)

NOTE: P values in the cohort sample are probabilities (two-tailed) obtained by using the normal approximation to the binomial for analysis of discordant pairs.[9]

[a] McNemar chi-square = 3.89, $p < .05$.

[b] McNemar chi-square = 2.23, $p > .10$.

According to the Health Belief Model, a media event can serve as a cue or a trigger for an individual to take a preventive health action but the effectiveness of the external cue depends on a complex host of variables including the perceived benefits of an action, the structural barriers to taking an action, as well as the intensity and duration of the cue.[4] On the basis of communication research, the rapid diminution of the effect of episodic information,[5–7] even if widely diffused (as with Mrs. Reagan's experience), may be overcome by community-based interventions[8] that maintain any changes in knowledge, attitudes, and behavior and that help in removing barriers to screening. The small increase in numbers of women contacting a health professional, and the direct attribution of the adoption of first-time use of mammography to the news of Nancy Reagan's breast cancer by a small number of women found in this study, may provide impetus for further work on the integration of such experiences with intervention programs to encourage preventive health behaviors.

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Is Testicular Cancer Incidence in Blacks Increasing?

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Abstract: Using data collected by the Surveillance, Epidemiology and End Results Program (SEER) from 1973–84, we found no evidence of an increase in the incidence of testicular cancer in Blacks, in contrast to an increase of nearly 33 percent in Whites. (*Am J Public Health* 1989; 79:1553–1554.)

Introduction

In 1985, Sagalowsky and Admire,[1] on the basis of treating four testicular cancer cases in Blacks in the early 1980s compared to none in the previous seven years, hypothesized that the incidence rate of testicular cancer was increasing in Blacks during the 1970s and early 80s. Epidemiologic studies in the past have reported no increase in testicular cancer in Blacks but definite increases in Whites.[2–6] We sought to update earlier studies.

Methods

Data were obtained from the Surveillance, Epidemiology and End Results (SEER) Program of the National Cancer Institute.[7] There were 4,788 cases of testicular cancer in Whites and Blacks identified between January 1, 1973 and December 31, 1984 by the SEER registries, of which greater than 99 percent were histologically confirmed. Testicular tumors were classified into three broad histologic categories using the International Classification of Diseases for Oncology (ICD-O)[8]: seminoma (9060-9064), non-seminoma germ cell (9070-9073, 9080-9083 and 9100-9102) and all other tumors. Population estimates based on US Bureau of the Census estimates were provided by the SEER Program. All rates presented are standardized to the age distribution of the US population in 1980 and expressed per million men per year. Poisson regression was used to evaluate whether the linear slope of the incidence rates by year of diagnosis differed from zero.[9]

Results

Table 1 shows the incidence rates of testicular cancer by year, age, histology and race during 1973–84. In Black men, with a total of 103 cases, there was no evidence of any increase in rate during the interval, for the individual histological categories, for all categories combined, or by age. In contrast, during the same time period, the incidence of testicular cancer in White men increased nearly 33 percent, primarily in age groups below 50 years and in seminomas.

Discussion

In Blacks, no increase in the incidence of testicular cancer was apparent during 1973–84. Data on testicular cancer in US Blacks for earlier decades are limited, and it

TABLE 1—Incidence* of Testicular Cancer in Blacks and Whites by Age and Histology: Surveillance, Epidemiology and End Results Program, 1973–84

	Time Period					
Characteristics	1973–75	1976–78	1979–81	1982–84	1973–84	Regression Coefficient (SE)**
Histologic Type†						
Black						
All	8.9 (19)††	10.4 (29)	9.0 (28)	8.3 (27)	9.2 (103)	−0.054 (0.088)
Seminoma	6.5 (13)	4.0 (11)	4.0 (12)	5.7 (17)	5.0 (53)	−0.066 (0.119)
Other Germ Cell	1.5 (4)	3.0 (9)	4.3 (14)	2.1 (8)	2.8 (35)	0.100 (0.163)
Other	0.9 (2)	3.4 (9)	0.7 (2)	0.5 (2)	1.4 (15)	−0.316 (0.231)
White						
All	38.3 (857)	42.0 (1087)	47.5 (1289)	51.1 (1452)	45.3 (4685)	0.098 (0.013)
Seminoma	18.7 (420)	20.0 (519)	22.6 (614)	25.9 (741)	22.0 (2294)	0.111 (0.019)
Other Germ Cell	15.9 (358)	17.3 (452)	19.3 (533)	19.9 (567)	18.2 (1910)	0.078 (0.021)
Other	3.7 (79)	4.8 (116)	5.5 (142)	5.4 (144)	4.9 (481)	0.112 (0.041)
Age (years)						
Black						
20–34	17.7 (10)	15.9 (12)	25.5 (22)	16.4 (16)	19.0 (60)	0.019 (0.119)
35–49	8.6 (3)	21.6 (9)	4.4 (2)	11.8 (6)	11.6 (20)	−0.081 (0.202)
50–64	10.8 (3)	6.1 (2)	5.9 (2)	5.8 (2)	7.0 (9)	−0.206 (0.305)
White						
20–34	77.7 (433)	86.2 (579)	104.1 (751)	107.3 (813)	95.1 (2576)	0.113 (0.018)
35–49	54.2 (211)	63.6 (274)	65.1 (293)	75.8 (362)	65.2 (1140)	0.103 (0.027)
50–64	34.6 (121)	28.0 (108)	24.8 (97)	35.5 (138)	30.6 (464)	0.014 (0.043)

*Rate per million men per year.
**Poisson regression coefficient and standard error for linear slope of incidence by year of diagnosis.
†Rates standardized to the age distribution of the 1980 US population.
††Number of cases shown in parentheses.

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remains uncertain whether there has been an increase over a longer time period. All of this contrasts to the clear steady rise of testicular cancer incidence in US White men, both during 1973–84 and earlier.

The explanation of this discrepancy in trends lies either in a difference in susceptibility to the agent(s) responsible for the increase seen in Whites and/or to a lower level of exposure to these agents. The similarity of the White:Black incidence ratio for testicular cancer in different populations supports the hypothesis that there are at least some racial differences in susceptibility. In South Africa's Cape district, the rate ratio was 3.3 (Whites compared to "coloured")[10] while the ratio in the US is around 4.5[4–6] Further, the incidence of testicular cancer in Blacks is very similar in different areas of the world.[11–15] However, the presence of such a difference in susceptibility between the races does not preclude the possibility that Whites have been increasingly exposed to a greater extent than Blacks in recent decades to some, as yet unidentified, exposure that predisposes to testicular cancer.

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Self-Report and Medical Record Report Agreement of Selected Medical Conditions in the Elderly

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Abstract: This study assessed agreement between self- and medical record report of medical conditions in an elderly population. Medical charts of 120 participants in a screening program were abstracted, and the questionnaire report of eight major conditions was compared with the medical record. There was substantial or moderate agreement between self-report and medical record report for each condition, although strength of agreement varied by condition. Self-report by elderly individuals compares favorably with medical record report of medical conditions. (*Am J Public Health* 1989; 79:1554–1556.)

Introduction

Information on medical conditions is frequently gathered by questionnaire because a standardized medical examination would be impractical and expensive, but the accuracy of self-reported information is seldom assessed. This is of particular concern in elderly persons, who may be more likely to misreport information than younger individuals. The purpose of this study is to evaluate the agreement between self-report and medical record report of selected medical conditions in elderly individuals.

Methods

Population

Individuals in this study have participated for at least three years in the Florida Geriatric Research Program, a health-screening program located in Dunedin, Florida. This free program is open to all ambulatory residents of Dunedin over 65 years of age; over 45 percent of the age-eligible residents have been enrolled. The program began in July 1975 and screens 2,200 persons each year.

Participants are seen annually for a clinic visit, where they are given a brief medical examination and complete a detailed questionnaire ascertaining previous and present illnesses. Detailed descriptions of this population have been published.[1]

For this analysis, a 10 percent random sample of participants who had their annual clinic visit between July 1, 1985 and December 31, 1985 was selected. These individuals

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were sent a letter requesting permission to review their medical records.

Medical Record Review

Mease Health Care, Inc. (MHC), a private hospital with a large outpatient clinic, serves the Dunedin community. All medical records (both inpatient and outpatient) of MHC were requested for sampled individuals. One of us (TLB) then reviewed each medical record and noted the occurrence and date of first diagnosis of the following conditions: angina pectoris, cancer, cataract, diabetes, fracture, hypertension, myocardial infarction, and stroke. Any non-definitive diagnosis (i.e., possible stroke) was also noted.

Questionnaire Responses

The wording of the questions used is presented in Table 1. Except for cataracts, possible responses to each question are: Yes, No, or Unknown. Possible responses to the question on cataracts are: One Eye, Both Eyes, or None.

Analysis

The medical record diagnosis for each condition (yes or no) was compared to the self-report (yes or no) obtained by questionnaires administered at all previous program visits. Medical record diagnoses after the date of completion of the questionnaires were ignored. We did not assume the medical record was more correct than the self-report. The percent agreement between the two reports, the kappa statistic, and z-scores for kappa were calculated.[2]

Results

In this study, 120 of the 122 randomly selected individuals consented to a medical chart review. The two participants not providing consent had died between their clinic visit and the request for permission to review. Of these 120 persons, 115 had received care at MHC and theoretically had charts available for evaluation; charts were located and reviewed for 68 women and 39 men (total = 107). At their first visit, participants ranged in age from 65 to 90 years ($\bar{x} = 71$ years). Of the 170 medical diagnoses noted in the medical records, 62 percent were diagnosed after 1975 and 38 percent prior to 1975.

The proportion with a positive response on medical record or self-report, the percent agreement between self-report and medical record report, and kappa are presented (Table 2). Angina pectoris, cataract, fracture, hypertension, and myocardial infarction were somewhat more likely to be noted in the self-report, whereas cancer and stroke were slightly more often noted in the medical record.

TABLE 1—Questionnaire Items Used in Self-Report of Selected Medical Conditions in the Elderly

Condition	Wording of Question
Angina	Have you ever been told by a doctor that you have angina?
Cancer	Have you ever been told that you had cancer and this was confirmed?
Cataract	Have you been told of cataracts in the *following*?
Diabetes	Do you have diabetes mellitus?
Fracture	Have you had any broken bones within the last five years?
Hypertension	Have you ever been told that you have hypertension or high blood pressure?
Myocardial Infarction	Have you had a heart attack, thrombosis, coronary, or myocardial infarction?
Stroke	Have you ever had a stroke?

TABLE 2—Percent Positive Responses by Self-Report and Medical Record Report, Percent Agreement Between Self-Report Medical Record Report and Kappa: Selected Conditions

	Positive Report by				
Condition	N	% Self	% Medical Record	% Agreement	Kappa
Angina	103*	26.2	15.5	85	.57
Cancer (Any)	107	24.3	31.8	89	.72
Cataracts	105*	50.5	34.3	76	.53
Diabetes	107	16.8	16.8	98	.93
Fracture	103*	22.3	13.6	91	.71
Hypertension	107	46.7	32.7	86	.71
Myocardial infarction	107	12.1	8.4	94	.70
Stroke	107	5.6	7.5	98	.85

*Four persons were excluded from these analyses either because a non-definitive diagnosis was listed in the medical chart or unknown was checked on the questionnaire.

The percent agreement between self-report and medical record report ranged from 76 percent for cataract to 98 percent for stroke and diabetes. The overall agreement was 89 percent. There were no differences in agreement by sex or recency of condition. All kappa values had p-values $<.001$.

Discussion

Public health researchers are concerned about the validity of self-reported information.[3,4] In this study, agreement is high between the self-report and medical record report of common medical conditions. Kappa scores for six of the eight conditions are >0.6, indicating substantial agreement.[5] Kappa values between .40 to .60, seen for the remaining two conditions, are evidence of moderate agreement.[5]

There are several limitations to this study. First, participants were not a representative sample, but volunteers who came to a screening program and may be different from non-participants in their interest and knowledge of health-related matters. Over 45 percent of the age-eligible residents of Dunedin were enrolled, however, and the self-reported prevalence of these conditions is very similar to the prevalence reported in three other community studies of elderly persons.[6]

A second limitation is that the respondents had all participated in this screening program, and participation may have affected their knowledge about their health status. However, elderly individuals in general are more knowledgeable about their health than are younger persons[7]; 80 percent of older Americans visit physicians at least once a year, with the average physician visits = 4.8/year.[8] Whether and to what degree participation in this program would enhance participants' knowledge over and above what they ascertain from physician visits is unknown.

An additional limitation is that the medical records were from the one major hospital/clinic in Dunedin. Participants may have had conditions diagnosed in other medical locations and this information may not be in the reviewed chart. The effect of this type of error would be to lower the agreement between the two sources.

Although overall agreement between self-report and medical record report is good to excellent, there is variability in the percent agreement among the medical conditions. This variability in prevalence by ascertainment source has been reported previously in younger persons,[4,9–13] and probably results from errors at each source. Self-reported information can be wrong for numerous reasons including a misunderstanding of the diagnosis presented, or the forgetfulness of the individual reporting. Likewise, medical records are not necessarily an accurate source of information.[14–21] Serious non-reporting or misreporting of diagnoses in medical charts has been found in several studies.[16–18,20]

Given the potential sources of error with both methods of ascertaining information, it is impossible to know which assessment comes closest to truth. For most important conditions this is probably not an issue, since self-report is significantly correlated with medical record report.

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Geographic Variation in the Occurrence of Hip Fractures among the Elderly White US Population

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Abstract: Geographic variations in hip fracture rates across the nine US Census Divisions were examined using data for elderly Whites from the 1979–85 National Hospital Discharge Survey. Rates varied considerably and were highest in the West North Central Division where rates for females were 50 percent higher than the US rate. Reasons for the geographic variations in hip fracture rates are unknown but do not appear to be closely related to latitude or fall-related deaths. (*Am J Public Health* 1989; 79:1556–1558.)

Introduction

Fractures of the hip result in over 250,000 hospital admissions each year and, in the elderly, represent 25 percent of all injury admissions.[1] A number of risk factors have been identified for osteoporotic hip fractures including increasing age, White race, and female sex.[2–5] Incidence rates for hip fracture also vary considerably from one country to another and a high rate has been reported for the United States in comparison with other countries.[2,3,6] This study explores geographic variations in hip fractures within the US using data from the National Hospital Discharge Survey (NHDS).

Methods

The NHDS collects data annually on a representative sample of discharges from nonfederal short-stay hospitals in the US.[1] The data set for this study consisted of all sample discharges from 1979 to 1985 among Whites, age 65 years or older in whom hip fracture (ICD-9-CM codes 820.0 to 820.9) was one of the discharge diagnoses. Blacks and other races were excluded from the analysis because of the small number of sample cases. Cases for which race was not stated were considered White based on previous demographic analysis.[4] All discharges whose hospitalization involved late effects of hip fracture (ICD-9-CM code 905.3) or complications of a previous fracture (ICD-9-CM code 733.8) or who were transferred to another short-stay hospital were excluded to minimize multiple counting of the same fracture. The final sample contained 6,701 discharges.

Geographic comparisons were limited to the nine US Census Divisions[7] because of the design characteristics of the

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NHDS.[8] Based on the sample, the projected total numbers of hip fracture patients discharged each year were computed by hospital Census Division using the NHDS weighting factors.[8] Age/sex-specific rates were derived from the mean annual number of discharges and Census estimates of the White civilian population as of July 1, 1982 for male and female age groups 65–74 years, 75–84 years, and 85 years and older. Age/sex-adjusted rates were based on the above age/sex-specific rates and the 1982 US White population as the standard. Standard errors were based on the 1982 NHDS,[9] were adjusted to account for aggregating multiple years of data,[8] and assumed the standard population was not subject to sampling error.

Results

Age/sex-adjusted hip fracture rates per 1,000 population and 95% confidence intervals are shown for the nine US Census Divisions and for the US in Figure 1. Comparisons of all pairs of rates among the nine Census Divisions using the Bonferroni Inequality[10] (comparison wise p = .0014) indicated the rate in the West North Central Division was significantly higher than the rates in all other Divisions except the Mountain and Pacific Divisions.

Age/sex-specific rates (Table 1) were generally higher in the West North Central Division for each age and for both sexes compared to other Divisions. For females, the West North Central rates were at least 45 percent higher than the corresponding US rates in each of the three age groups. Only for males age 75–84 were discharge rates higher in other Divisions (Mountain and Pacific). The highest rates also occurred in the West North Central Division in each of the seven years from 1979 to 1985, the time period over which the data were aggregated.

Discussion

Although hip fractures typically result in hospitalization, hospital discharges cannot be assumed to equal hip fracture incidence—particularly because of multiple counting of cases due to transfers from one hospital to another, and readmissions for additional treatment of old fractures.[11–13] To minimize this potential bias, all transfers to another short-stay hospital and all hip fracture cases with a secondary diagnosis of malunion or late effects were excluded. The data base does contain refracture cases which may represent as many as 15 percent of the discharges.[5,14–16] However, there is no reason to believe that refracture rates would vary greatly by geographic area and, as new events, should be included in estimates of fracture incidence.

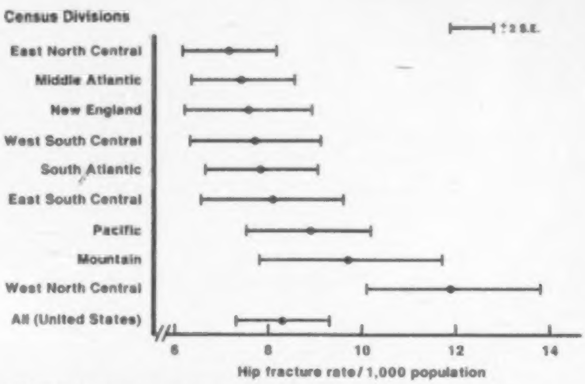

FIGURE 1—Mean Annual Hospital Discharge Rates for Hip Fractures per 1,000 White, Elderly, Civilian Population by Census Division
New England: ME, NH, VT, MA, RI, CT
Middle Atlantic: NY, NJ, PA
South Atlantic: DE, MD, DC, WV, VA, NC, SC, GA, FL
East North Central: MI, WI, IL, IN, OH
East South Central: KY, TN, MS, AL
West North Central: MN, ND, SD, NE, IA, MO, KS
West South Central: AR, OK, LA, TX
Mountain: MY, WY, ID, CO, UT, NV, NM, AZ
Pacific: WA, OR, CA, AK, HI

Factors such as listing old hip fractures as secondary diagnoses, admitting patients with old hip fractures for arthroplasty, and admitting patients residing outside the Census Division of the hospitals could vary by Census Division and thus are potential sources of bias. Analyses of these variables showed little geographic variation, however.

To corroborate the findings using a different data source, discharge data from a 20 percent sample of all inpatient bills were obtained from the 1985 Health Care Financing Administration (HCFA) MEDPAR file. Hip fracture rates for aged Medicare beneficiaries were also highest in the West North Central Division compared to other Divisions.

This study indicates that discharge rates for hip fracture in elderly Whites is higher in the West North Central region of the US than in other Census Divisions for reasons which are not clear. Several factors have been associated with an increased risk of osteoporotic fractures[2,3,5,16–19] but many, such as diet and exercise, would seem less prevalent in the farm states of this

TABLE 1—Hip Fracture Rates per 1,000 Elderly, White Persons and Standard Errors ( ) by Age, Sex and Census Division

	Age (years)							
	Males				Females			
Census Division	65 & older	65–74	75–84	85 & older	65 & older	65–74	75–84	85 & older
New England	4.1(0.7)*	1.9(0.5)	5.6(1.4)	15.8(4.9)	9.7(1.0)*	3.6(0.8)	12.1(2.1)	36.0(6.2)
Middle Atlantic	4.4(0.5)*	1.7(0.4)	6.3(1.2)	18.3(4.0)	9.3(0.8)*	4.0(0.6)	12.4(1.7)	29.5(4.4)
East North Central	4.2(0.5)*	1.6(0.4)	5.6(1.1)	19.9(4.0)	8.9(0.8)*	3.5(0.6)	11.4(1.6)	30.1(4.4)
West North Central	6.0(0.8)*	2.9(0.7)	6.8(1.6)	27.0(5.7)	15.5(1.4)*	6.2(1.1)	20.7(3.0)	51.0(7.8)
South Atlantic	4.2(0.5)*	1.3(0.3)	5.4(1.1)	22.6(4.8)	10.0(0.9)*	3.5(0.6)	13.7(1.9)	34.5(5.3)
East South Central	4.7(0.8)*	2.0(0.6)	5.8(1.6)	22.2(6.4)	10.1(1.2)*	4.2(0.9)	14.7(2.6)	27.9(5.8)
West South Central	4.8(0.7)*	1.6(0.4)	7.1(1.7)	20.8(5.3)	9.5(1.0)*	2.5(0.6)	12.8(2.1)	38.3(6.5)
Mountain	5.6(0.9)*	2.4(0.7)	8.2(2.1)	21.1(6.5)	12.2(1.4)*	4.7(1.0)	15.9(3.0)	42.6(8.4)
Pacific	4.5(0.6)*	1.9(0.4)	8.2(1.6)	10.6(2.9)	11.5(1.0)*	4.3(0.7)	16.1(2.3)	36.6(5.5)
All (US)	4.8(0.5)	2.2(0.2)	6.4(0.5)	19.6(1.7)	10.4(0.9)	3.9(0.3)	14.0(0.9)	35.2(2.3)

*Age-adjusted.

region. Sunlight exposure is unlikely to be a major factor because rates are dramatically different among regions at similar latitudes (e.g., West North Central and East North Central Divisions). Further, a relationship of geographic variation in hip fracture rates to reported mortality from falls is not evident despite the fact that falls account for most hip fractures in the elderly.[20,21] All seven States in the West North Central Division have above-average death rates from falls in elderly Whites, and Iowa and Minnesota are among the 10 States with the highest rates.[22] However, New England has a high death rate for falls and a relatively low discharge rate for hip fractures. (If case-fatality rates for hip fracture are relatively constant, differences between discharge and mortality rates may reflect geographic variations in the reporting of falls as a cause of death on the death certificate.[23,24]) It is interesting that a relatively high proportion of residents in the West North Central region originate from Scandinavian countries[25] where the incidence of hip fractures is also high.[2,3,6] Further investigation of the factors responsible for the geographic variations observed may lead to new insights into the causes of hip fractures.

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T4

Lead Exposure and Radiator Repair Work

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Abstract: In 1986, the ambient air for lead in radiator repair shops in the Minneapolis-St. Paul metropolitan area exceeded the Occupational Safety and Health Administration (OSHA) action level in nine of 12 shops sampled by Minnesota OSHA. We therefore sought to determine the prevalence of lead exposure/toxicity in this industry. Thirty-five radiator shops were identified, 30 were visited, and 53 workers were studied. The mean blood lead level was 1.53 (range 0.24–2.80). Seventeen individuals had blood lead levels ≥1.93 μmol/L (40 μg/dl). The mean zinc protoporphyrin level (ZPP) was 0.55 μmol/L (range 0.16–1.43). No single worksite or personal characteristic was a strong determinant of either blood lead or ZPP level. (*Am J Public Health* 1989; 79:1558–1560.)

Introduction

Workers in the automotive radiator repair industry are known to be at risk for exposure to inorganic lead from lead-based solder used in the repair of radiators.[1–8] Lead fumes arise from molten lead and lead oxide dusts result from grinding and wire brushing radiator joints. These operations are done less frequently now than in the past, largely due to the recognition of the health hazards they cause.[9] It is claimed that more efficient ventilation, and self-contained breathing apparatus for radiator cleaning have reduced much of the lead exposure.

Results of recent studies in the upper Midwest area of the United States revealed that radiator repair shops may have elevated air lead levels, and that repair workers may have

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elevated blood lead levels, occasionally higher than 2.90 μmol/L (60 μg/dl).[2–5] An auto radiator trade journal performed a questionnaire survey in 1977,[6] in which 100 radiator workers voluntarily reported information including recent blood lead levels, work history and habits, shop characteristics, and personal health history. Blood lead levels ranged from 0.63 to 5.45 μmol/L. Departments of health in some states have attempted to study auto radiator repair shops in a systematic way, with varying degrees of success. Although no publications in the scientific literature resulted, blood lead levels in excess of 4.34 μmol/L were found in some workers (personal communication, Michigan and Wisconsin Departments of Health).

In the summer of 1986, Minnesota OSHA (Occupational Safety and Health Administration) sampled the ambient air for lead in nine radiator repair shops in the Minneapolis-St. Paul area. Air in two of these shops exceeded three times the OSHA standard for ambient lead, and three others exceeded the OSHA action level. The standard Permissible Exposure Limit for lead for a 40-hour work week is 0.05 mg/m^3. This information, together with the results of previous studies, led us to question the degree of lead exposure and toxicity in auto radiator repair workers in the Minneapolis-St. Paul area. We sought to determine the prevalence of lead exposure and toxicity in these workers and characteristics associated with elevated lead levels.

Methods

All automobile radiator repair shops within Minneapolis-St. Paul and their closest suburbs were identified using the Minneapolis-St. Paul yellow page directories. The owners were asked to permit a visit by a study investigator to administer a questionnaire to shop employees and obtain blood samples for lead and zinc protoprophyrin levels (ZPP).

During November 1986, a visit was made to each shop whose owner consented to participate in the study. The shop owner or manager was interviewed to obtain information on shop size, number of cars serviced, and workplace conditions. Permission was then obtained to interview all radiator repair workers present at that time. Each radiator repair worker was then asked demographic questions, recent work history, workplace and general health practices, directed health history, and whether the worker had children under the age of six living in the home. If such children were present, permission was requested to screen them for lead.

Blood lead determinations were done by the anodic stripping voltametry method.[7] ZPP determinations were done at the University of Minnesota using a hematofluorometer.[8] Individual workers were each informed of their blood lead levels by letter, with an accompanying explanation of the results. Shop owners were also furnished with this information if the employee had given written consent to do so.

Later, the shops were visited by an individual hygienist who took area samples for three to six hours using high flow air sampling pumps.[10] Personal breathing zone air samples were also done by attaching the filter cassette of the sampler to the shirt lapel of the participating worker. Analyses of samples were done by atomic absorption.[11] Each shop owner was informed of air measurements with an explanation of the level of lead in the air.

Statistical analyses were done using the Statistical Package for the Social Sciences (SPSS). Shop data for individuals from the same shops were identical, but were treated independently for purposes of analysis because the shops were small, with most having one or two workers, and analysis of covariance showed neither shop size nor number of radiators repaired per year in a specific shop to be significant covariates.

Regression analyses were done with blood lead and ZPP as dependent variables, and several of the items from the shop and individual questionnaires, and area and personal air lead samples, as independent variables. All specified variables were included in the regression model.[12,13] A chi square statistic was computed for categorical variables. In the latter analyses, blood lead (1.93 and $\geq$1.93 μmol/L) and ZPP ($<$0.89 and $\geq$0.89 μmol/L) were treated as categorical variables with two levels each.

Results

Shop Characteristics

Thirty-five radiator shops were identified in the metropolitan area. Of these, 30 were visited, making the shop participation rate 85.7 percent. One shop was not visited because, although the shop owner was cooperative, the radiator repair workers themselves refused to participate. The owners of the other four shops refused to participate.

Shops were small, usually 300 to 600 square feet, and there was generally no interior finish (Table 1). The amount of business varied greatly between shops. While use of specific work site ventilation was often reported by shop owners and the individual workers, only a few had local ventilation and most ventilation systems were not in operation. All but one shop supplied tools for their employees, all had salaried employees, and none had working showers. All shops used solder comprised of 60 percent lead and 40 percent tin.

Area and personal air sampling for lead levels were done in 24 of the 30 shops. Six shops refused to have air monitoring. Personal air lead samples were obtained from individuals in 16 of the 24 shops. Workers refused to wear a pump in eight shops. The mean area lead value was 0.040 mg/m^3 with a range of 0 to 0.281 mg/m^3 (SD = .060). The mean personal air lead value was 0.113 mg/m^3 with a range of 0 to 0.590 mg/m^3 (SD = .020).

Worker Characteristics

A total of 57 radiator repair workers were identified. Of these, four refused to participate in the study, but at least one radiator repair worker was evaluated in each participating shop. Of these workers, the mean age was 37.1 years (SD = 2.8 years), mean education was 11.9 years (SD = 2.8 years), and mean work experience was 14.3 years (SD = 11.2 years). Table 2 details other worker characteristics. Most of the workers identified job duties other than radiator repair that they normally performed, but none involved significant lead exposure. No worker reported ever having been treated for lead toxicity, although five out of 53 reported having had elevated blood lead levels in the past.

TABLE 1—Characteristics of Thirty Radiator Repair Shops

Variables	Mean	Standard Deviation	Range
Age of Shop (years)	17.0	15.5	<1–50
Number of Employees	3.6	2.7	1–10
Number of Radiator Repair Workers	1.9	1.5	1–8
Number of Cars/Trucks Repaired Per Week	43.2	40.8	0–200
Number of Radiators Repaired Per Week	35.5	38.2	1–200
Number of Radiators Repaired Per Year	1281.7	932.2	50–3500

TABLE 2—Self-reported Characteristics of Radiator Repair Workers

Characteristics	Category	Number (%)
Respiratory Protection	Never	41 (77.4)
	≤25% of time	8 (15.1)
	>25% of time	4 (7.5)
Gloves Used	Never	7 (13.2)
	≤50% of time	8 (15.1)
	>50% of time	38 (71.7)
Eat Lunch at Work Stations	Never	48 (90.6)
	≤25% of time	1 (1.9)
	>25% of time	4 (7.5)
Smoke Cigarettes	Yes	32 (60.4)
	No	21 (39.6)

Twelve individuals reported having children under the age of six living at home. Sixteen children were later screened for blood lead and, with one exception, levels were below 0.724 μmol/L, and a mean of 0.45 μmol/L (range = 0.24 to 1.06 μmol/L). This did not correlate well with workers' blood leads, and no further analyses were done.

As seen in Tables 3 and 4, there was a wide range of blood lead levels among the workers. Symptoms associated with lead toxicity included frequent headaches, abdominal cramps, constipation, and muscle aches. None of these symptoms was common in the workers but none was significantly associated with either blood lead or ZPP level.

In bivariate analyses, two variables were associated with increased blood lead levels: number of radiators repaired in the past month (0/8 vs 17/44; 95% CI = 0.09, 0.69)*; and shop age (4/24 vs 13/29; 95% CI = 0.21, 0.35). Years of work experience was the only variable found to be related to ZPP level (3/20 vs 18/33; 95% CI = 0.12, 0.67). Confidence intervals are for the difference in the proportion of workers with elevated blood lead in the high-risk (exposure) group compared to the proportion of workers with increased blood lead in the low-risk (exposure) group.

Multiple regression was used to examine the simultaneous effects of worksite and personal factors. Four variables were entered into the regression for blood lead level: years of work experience ($\hat{B}$ = 0.25, SE 0.15), worker time spent in radiator repair during the last month (2.00, SE 1.43) and last year (0.03, SE 0.18), and shop age (5.97, SE 744), the total r^2 was 0.25. Work experience was the only variable that contributed to the ZPP level (0.89, SE 0.18, r^2 = 0.34).

TABLE 3—Number of Individuals with Blood Lead Levels by Approximate Quartiles*

Blood Lead Level (μmol/L)	Number of Workers
>1.93	15
>1.50–1.93	11**
>1.06–1.50	13
0.24–1.06	1[illegible]

Mean = 1.53, Standard Deviation = .61
**Two workers in this group had a blood level of 1.93 μmol/L

TABLE 4—Number of Individuals with Free Erythrocyte Protoporphyrin Levels by Approximate Quartile*

FEP Level (μmol/L)	Number of Workers
>.067	15
>0.41–0.67	12
>0.30–0.41	12
0.16–0.30	13

Mean = 0.53, Standard Deviation = 0.31

Discussion

About one-third of the workers had lead levels that were elevated by OSHA standards.[14] Minnesota radiator repair workers had lead levels that were similar to those found in recent studies in this country.[1–5,15] These levels are considerably less than those routinely seen in the high-risk occupations such as lead smelting, storage battery manufacturing, or metal foundries were levels occasionally exceed 4.83 μmol/L.[16] However, the blood lead level[3] found in radiator workers exceeds those in the general population, where mean lead levels in adult males is 0.63–0.72 μmol/L.[16] Auto radiator repair workers appear to be at a risk of mild to moderate elevations in blood lead levels.

No single worker characteristic or practice was a good predictor of elevated blood lead. Only two shop characteristics—volume of radiator repair done and shop age—were associated with elevated blood lead levels. Environmental data did not correlate well with blood lead levels. This may have occurred because of the variability of the volume of work in each shop from day to day as well as our having been invited into shops on "less busy days." The data presented here confirm earlier studies of this industry. There is need for worker education, safer worker practices, and better shop design and ventilation in the radiator repair industry.

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*One worker had not done radiator repair during most of the month prior to the study.

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Acute Illnesses among Los Angeles County Lifeguards According to Worksite Exposures

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Abstract: Due to public concern regarding the potential adverse health effects of exposure to Santa Monica Bay waters, a case-control study of acute illnesses among Los Angeles County lifeguards was conducted. Workers' compensation claimants (N = 112) were matched to healthy lifeguards working in the same year and having the same job classification. Conditional logistic regression showed that the southernmost worksites of the bay were associated with all acute illnesses (OR = 14.4, 95% CI = 4.7, 44.8) and with ear infections (OR = 12.5, 95% CI = 2.9, 53.4). (*Am J Public Health* 1989; 79:1561–1563.)

Introduction

The quality of water in Santa Monica Bay of Los Angeles County, California has been the subject of debate because of the dumping of industrial waste and sewage into the bay and untreated discharges into the surf zone from numerous storm drains along the shore.

Most studies of water quality and adverse health outcomes have established a direct correlation between pathogen levels in the water and disease incidence in swimmers,[1–5] while two found no relationship.[6,7] Cabelli and coworkers found a dose-response relation between swimming-associated gastrointestinal symptoms and water quality (as measured by enterococcus concentrations) at a group of New York City beaches.[1] In a study of five Ontario lakes conducted by Seyfried and Cook,[4] otitis externa was related to water levels of *Pseudomonas aeruginosa* and to the frequency of swimming.

To assess the potential adverse health effects of exposure to the waters of Santa Monica Bay, we studied work-related acute illnesses in an occupationally exposed population—Los Angeles County lifeguards. This is the first study of a defined population exposed to these waters.

Methods

We used a case-control design, with cases defined as all episodes in which lifeguards made a compensable claim for a work-related, acute illness in the years 1980 through 1986. Lifeguards with repeated acute illness claims in a single year were included only once. A healthy lifeguard was chosen as a control for each case. The control lifeguard worked the same year that the case was ill, had no acute illness claim in that year, and worked in the same job class (i.e., temporary assignment, permanent assignment, lieutenant, etc.).

For each case and control, the daily work assignment logs for the month including the case's date of illness were searched. Details of the work assignments on the case's date of illness and seven days prior to that were recorded, including section of beach, tower, and hours at each tower.

Descriptive analyses used SAS computer programs,[8] and conditional logistic regression estimates[9] used BMDPLR.[10]

Results

During the seven-year study period, 112 acute compensable illnesses occurred among the Los Angeles County lifeguards. The peak year was 1980 with 28 claims, while 1985 and 1986 had only eight claims each. Most claims were due to otitis externa and unspecified ear infections (N = 69). Other diagnostic categories were otitis media (N = 15), dermatitis (N = 6), rash (N = 2), skin infection (N = 6), and conjunctivitis (N = 5).

The Department of Beaches and Harbors of Los Angeles County has divided the county beaches into three sections for administrative purposes (Figure 1): Southern, Central, and Northern. The age-adjusted odds ratios (ORs) for working at each section of beach compared with not working at all (Table 1) showed a very strong association between working Southern section beaches and acute illness. Similar associations were observed with illness restricted to otitis externa or unspecified ear infections (OR for Southern section = 12.5, 95% CI = 2.9, 53.4). Ten cases and 41 controls did not work at all during the reference week. The odds ratio for working as a Los Angeles County lifeguard at any location was 8.3.

Since ear infections are related to the amount of time spent in the water,[7,11] we also analyzed the effect of beach location using only those pairs where both subjects worked at all during the reference week (N = 64) with working as a proxy for swimming. The southernmost beaches within the bay proper were most strongly associated with acute illness. To remove the effect of 13 repeated ear infections occurring in different years to some "disease-prone" individuals, we

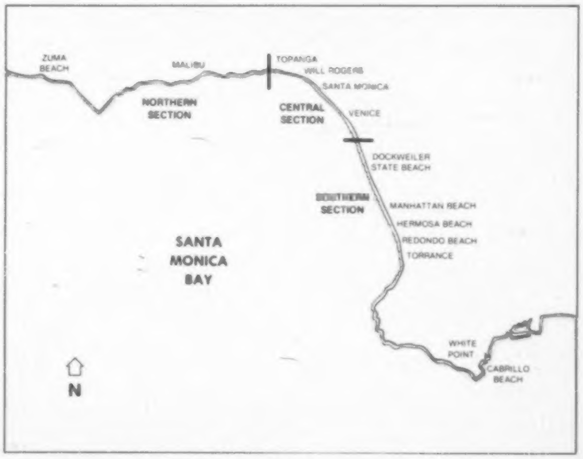

FIGURE 1—Los Angeles County Beaches

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TABLE 1—Age-Adjusted Conditional Disease Odds Ratios for Working at Various Beaches

Beach	Odds Ratio (95% CI)
All beaches combined	8.3 (2.9, 23.5)
Southern section beaches combined	14.4 (4.7, 44.8)
Cabrillo Beach-White Point	5.4 (0.8, 36.2)
Torrance-Redondo Beach	17.0 (2.9, 100.2)
Hermosa Beach	36.4 (6.7, 198.2)
Manhattan Beach	15.7 (2.8, 89.7)
Dockweiler State Beach	15.6 (2.2, 111.0)
Central section beaches combined	5.3 (1.8, 15.5)
Venice	10.7 (2.0, 57.2)
Santa Monica	7.0 (1.9, 24.6)
Will Rogers	6.4 (0.4, 103.8)
Northern section beaches combined	4.4 (1.4, 14.1)

repeated the analysis excluding these episodes. The odds ratios were diminished slightly but again the southernmost beaches within the bay were most strongly and significantly associated with illness.

Discussion

All illnesses studied for this report were compensated through Los Angeles County's workers' compensation system, and were therefore physician-diagnosed. Almost certainly less misclassification occurs with such data than with self-reports of symptoms.[2,6,12,13] On the other hand, workers' compensation reports probably yielded an underestimate of the total number of work-related illness events. It is possible that lifeguards who experienced minor symptoms did not seek medical attention or might not have associated symptoms with their jobs. This may explain the absence of gastrointestinal illnesses.

Although it is possible that lifeguards working at certain beaches were more likely to report acute illness, we know of no specific work pattern that would create such a bias. Also we noted a high odds ratio in Venice Beach in the Central section, which is administratively distinct from the Southern section.

We lacked data to control for other water exposures, e.g., swim workouts at the worksite or recreational exposure to the bay at the worksite or other locations. We had no data on swimming habits, such as head submersion, associated with ear problems.[11,13] Matching on job class partly controlled for differences in job tasks which could affect the subjects' amount of exposure to water while at work. For example, temporary lifeguards cover most of the tower assignments and therefore may have been required to enter the water more often, on average.

The reason for the decline in work-related acute illnesses over the study period is not known. Lifeguards may have begun mitigating their risk of illnesses by reducing their water exposures or by changing swimming habits.

A recent report on Santa Monica Bay indicated that storm drains and creeks are responsible for the levels of the indicator bacteria (enterococci and coliforms) measured at shoreline. Whether these bacteria are indicative of human pathogenic agents is unclear.[14] Using maps provided by the Los Angeles County Department of Public Works, we calculated the number of storm drains (500 feet or longer) or creeks per 10,000 linear feet of coastline (Table 2). These ratios were generally higher in Southern beaches, except for Dockweiler State Beach; but that beach includes the outlet for the largest drain into the bay which may contaminate a wider area than most drains. A recent raw sewage spill into this drain resulted in beach closures which included Venice and Dockweiler beaches.[15] We present this ecologic association as one possible explanation for our study results, recognizing that microbial contamination of Santa Monica Bay is a complex, multifactorial, and only partially understood process.

The findings of this study, the first of its kind, suggest that acute illnesses among lifeguards were associated with Southern Santa Monica Bay work locations.

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TABLE 2—Storm Drain and Creek Outlets into Santa Monica Bay and other Los Angeles County Beaches

Beach Areas	Number of Storm Drains and Creeks per 10,000 Feet of Coastline	Characteristics of Catchment Area	Comments
Southern Section			
Cabrillo Beach-White Point	7.2	Residential and commercial	Los Angeles Harbor ship channel empties east of Cabrillo Beach
Torrance-Redondo Beach	7.9	Residential and some commercial	Narrow catchment area; mostly winter rain season flow
Hermosa Beach	9.0	Residential and some commercial	Narrow catchment area; mostly winter rain season flow
Manhattan Beach	5.5	Residential and some commercial	Narrow catchment area; mostly winter rain season flow
Dockweiler State Beach	2.2	Residential, commercial, and heavy industrial	Ballona Creek at Northern edge of beach drains extremely large area and occasionally drains raw sewage spills
Central Section			
Venice	3.1	Residential and commercial	Moderate catchment area
Santa Monica	4.4	Residential, commercial, and industrial	Moderate to large catchment area
Will Rogers	4.4	Residential and unimproved parkland	Moderate to large catchment area
Northern Section	2.9	Sparse residential and parkland	Large catchment area; some raw sewage contamination from septic tanks

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